

**TO STUDY THE CHILDHOOD SEIZURES AND INCIDENCE OF
FINDINGS IN EEG AND NEURO IMAGING AT
TIRUNELVELI MEDICAL COLLEGE**

Dissertation submitted in partial fulfilment of the

Requirement for the award of the Degree of

M.D. DEGREE – BRANCH VII

PAEDIATRICS

APRIL 2016

TIRUNELVELI MEDICAL COLLEGE HOSPITAL



THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY,

CHENNAI,

TAMIL NADU

CERTIFICATE

This is to certify that the Dissertation entitled “**TO STUDY THE CHILDHOOD SEIZURES AND INCIDENCE OF FINDINGS IN EEG AND NEURO IMAGING AT TIRUNELVELI MEDICAL COLLEGE**” submitted by Dr.P.Babu Balachandar, MBBS., DCH., to The Tamilnadu Dr.M.G.R. Medical University, Chennai, in partial fulfilment for the award of M.D (Paediatrics) is a bonafide work carried out by her under my guidance and supervision during the academic year 2014-2016. This dissertation partially or fully has not been submitted for any other degree or diploma of this university or other.

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DECLARATION

I, Dr.P.Babu Balachandar, MBBS., DCH., solemnly declare that the Dissertation titled **“TO STUDY THE CHILDHOOD SEIZURES AND INCIDENCE OF FINDINGS IN EEG AND NEURO IMAGING AT TIRUNELVELI MEDICAL COLLEGE”** had been prepared by me under the expert guidance and supervision of **Prof.Dr.C.Krishnamurthy,MD.,(Paediatrics),**Professor, Department of Paediatrics, Tirunelveli Medical College Hospital, Tirunelveli.

The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the regulation for the award of M.D. Degree (Branch VII) in Paediatrics.

It was not submitted to the award of any degree/diploma to any University either in part or in full previously.

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ACKNOWLEDGEMENT

I am very much thankful to the Dean **Dr.K.Sithy Athiya Munarvah**, Tirunelveli Medical College Hospital, Tirunelveli, who has granted permission to do this study in this institution,

I take this opportunity to express my deepest sense of gratitude to professor **Prof.Dr.C.Krishnamurthy,MD.,(Paediatrics).**, Head of the Department of Paediatrics, Tirunelveli Medical College Hospital, Tirunelveli for encouraging me and rendering timely suggestions and guiding me throughout the course of this study. I will be forever indebted to her for her constant support.

I sincerely thank my professor **Dr.T.R.R. Ananth Shri, M.D.,(Paediatrics).**, **Dr.M.Mathivanan,M.D.,(DCH).**, **Prof.Dr.A.S.Babu Kandakumar, MD.,DCH.**, for their support and guidance.

I am very much thankful to professor **Dr.Nellaiappar.M.D.(Radiology)** Head of Department of Radiology for providing valuable support and guiding through the study.

I am very much thankful to professor **Dr.Radha D.M.(Neurology)** Head of Department of Neurology for providing valuable support and guiding through the study.

I am extremely thankful to my guide.,
Prof.Dr.C.Krishnamurthy,MD.,(Paediatrics)., for guiding me throughout the study.

I am extremely thankful to all my **Assistant Professors** of the **Department of Paediatrics** for their guidance and support throughout my study period in this institution.

I thank Mr. **P. Arumugam** statistician for their useful inputs.

I wish to express my gratitude to my parents, my brother, my husband and my son & daughter for their support throughout my study.

I also like to express my gratitude to my friends and colleagues who have always been a source of love, support and encouragement.

Last but not least, I am very much thankful to all antenatal mothers of Tirunelveli Medical College without whom this study would not have been possible.

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TO STUDY THE CHILDHOOD SEIZURES AND INCIDENCE OF FINDINGS IN EEG

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INTRODUCTION

A seizure is a transient occurrence of symptoms and signs resulting from abnormal synchronous or excessive neuronal activity in the brain.

About 5 % of children experience convulsion during the first five years.

A good description of seizures include mode of onset , details of aura,

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ABSTRACT

BACKGROUND

To study the childhood seizures and incidence of findings in eeg and neuro imaging at tirunelveli medical college and find out any correlation between the two investigation in seizures.

PURPOSE

The study is conducted to find the significance of EEG and neuro imaging in the unprovoked afebrile seizures. And to study the partial seizures and its EEG and neuroimaging findingsThe other type of seizures like meningitis,trauma congenital anamoly has a known etiology for the seizures. So these types are excluded in the study.

METHODS

A total of 50 children were included in the study with unprovoked afebrile seizures admitted in Pediatric department of Tirunelveli medical college, Tirunelveli. All children who satisfied the inclusion criteria were included in the study. The data regarding their name, age,sex, , type of seizures (according to international classification of epileptic seizures), past history of seizures, , drug history, development history, family history are collected in a preformed proforma (Annexure). The examination

findings are recorded. EEG recording, CT scan / MRI scan are done .

Findings in them are recorded.

RESULTS

Among the 50 children with unprovoked afebrile seizures, 70% of patient had generalised seizures and 30% patient had partial seizures.

82 % had EEG abnormality. Abnormal Neuroimaging is found in 18% of the patient. Majority of the abnormal Neuroimaging is shared by partial seizures group.

CONCLUSIONS

82% the children with seizures had abnormal Electroencephalogram.

73.1% had shared by generalised seizures and 26.9 % shared by partial seizures.

Higher incidence of neuroimaging abnormality is seen partial seizures with abnormal Electroencephalogram

There is higher correlation between neuroimaging abnormality and focal Electroencephalogram changes in partial seizures when compared to generalised seizures..

Electroencephalogram and Neuroimaging are mandatory in evaluating Children with afebrile seizures.

KEYWORDS

Electroencephalogram, Computed tomography, magnetic resonance imaging.

INTRODUCTION

A seizure is a transient occurrence of symptoms and signs resulting from abnormal synchronous or excessive neuronal activity in the brain.

About 5 % of children experience convulsion during the first five years.

A good description of seizures include mode of onset , details of aura, type of seizures, automatism, associated behavioural abnormalities, postictal phase should recorded. An accurate description is more informative than detailed examination and investigation. Perinatal, developmental history, family history of seizures will help in determining the cause.

Several times a child may present with condition that mimic seizures and misinterpreted as seizures. These include Breath holding Cough syncope, Narcolepsy, Night terror, Tics.

The Epileptic seizures are classified by

THE INTERNATIONAL CLASSIFICATION OF EPILEPTIC SEIZURES ¹

Into two categories

1. Generalised seizures
2. Focal or partial seizures

GENERALISED SEIZURES-

First clinical and EEG changes indicates synchronous electrical activity of both hemispheres

PARTIAL SEIZURES-

First Clinical and EEG changes suggests initial activation of a system of neurons limited to one part of hemisphere

CLASSIFICATION OF SEIZURES¹

A-SELF LIMITING SEIZURES TYPE

GENERALISED SEIZURES

1. Tonic
2. Clonic
3. Tonic Clonic
4. Myoclonic
5. Atonic
6. Absence

Typical

Atypical

Myoclonic

PARTIAL SEIZURES

1. Simple Partial

(No impairment of consciousness)

- Motor

- Sensory

- Autonomic

- Psychic

2. Complex Partial seizures

(Consciousness impaired)

- Simple partial seizures followed by loss of consciousness

- Simple partial seizures with impaired consciousness at onset

- Partial seizures evolving to secondary generalised seizures

B-CONTINUOUS SEIZURES TYPES

1. FOCAL STATUS EPILEPTICUS

Epilepsia partialis continua of Kojevnikov

Aura continua

Limbic status epilepticus

2. GENERALISED STATUS EPILEPTICUS

Generalized tonic-clonic status epilepticus

Clonic status epilepticus

Tonic status epilepticus

Absence status epilepticus

Myoclonic status epilepticus

EPILEPSY¹

is a disorder of the brain characterised by neurobiological, psychological, cognitive, and social consequences of the condition by a predisposition to generate seizures

To diagnose Epilepsy usually requires at least one occurrence of unprovoked seizures and subsequent similar seizures with similar or characteristic EEG and clinical findings to convincingly demonstrate and enduring predisposition to develop recurrence.

Epilepsy is to be considered when two or more unprovoked seizures occurring in a time more than twenty four hours.

ACUTE SYMPTOMATIC SEIZURES

Is a general term where the seizures occurs secondary to a brain insult due to meningitis or electrolyte imbalance

SEIZURE DISORDER

Includes any one of the several disorder including epilepsy, febrile seizures, single seizures and acute symptomatic seizures

EPILEPTIC SYNDROME

Is a disorder that manifest one or more specific seizures types and has a specific age of onset and specific prognosis.

SYMPTOMATIC EPILEPSY

Is caused by underlying brain disorder.(e.g. epilepsy secondary to tuberous sclerosis)

IDIOPATHIC EPILEPSY

Is a disorder where there is no underlying disorder affecting the development or neurological function

GENERALISED SEIZURES AND RELATED EPILEPTIC SYNDROME¹

TONIC CLONIC SEIZURES

It is the most frequent form of childhood epilepsy. It has four phases.

1. Aura
2. Tonic
3. Clonic
4. Postictal phase

Aura is a transitory premonitory symptom that occurs at the onset of seizures. *Aura* can be sensory, visceral, motor, autonomic

Tonic phase has skeletal muscle goes in sustained contraction. Muscle rigidity is more seen in the antigravity muscles. Child will have loss of consciousness, upward gaze, frothing of mouth. Involuntarily will pass urine and stool.

Clonic phase has rhythmic alternating contraction of a group of muscles.

Post ictal phase will have little recollection of the events occurred

First aid procedures should be followed for all GTCS patient includes airway breathing circulation, positioning the patient to sideways, positioning the head and clearing the airway.

PETIT MAL EPILEPSY¹

Patients may outgrow before adulthood and it starts in the mid childhood . $\frac{1}{4}$ of the patients will have GTCS.

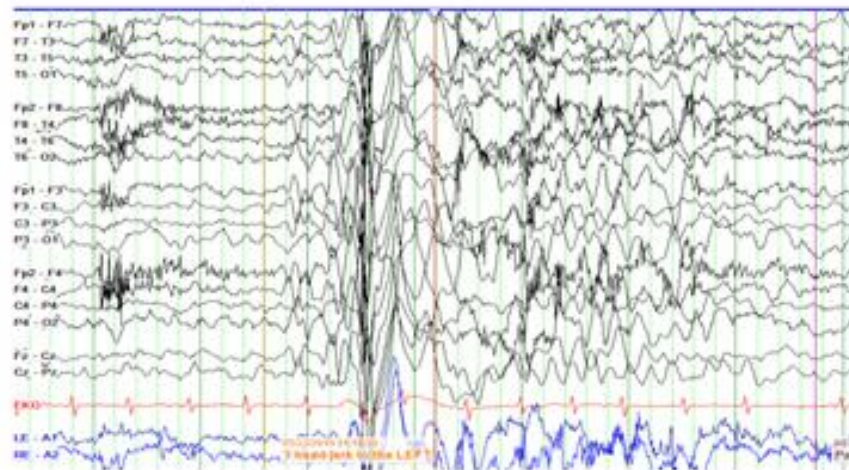
BENIGN MYOCLONIC EPILEPSY OF INFANCY ¹

Has generalised 3Hz spike and wave discharge onset of myoclonic epilepsy as early as 1st year of life.

Normal EEG Awake



Myoclonic Seizure



FEBRILE SEIZURES PLUS SYNDROME

Present with febrile seizures and multiple type of generalised seizures runs in many family members. Different febrile seizures and generalised seizures runs in different individuals in same family.

WEST SYNDROME

Consists of triad of development regression, classical hypsarrythmia in EEG and infantile Spasm. It is characterized by sudden drop attack often termed as salaam spells.

Hypsarrythmia in EEG shows slow high voltage multifocal spikes with chaotic background.

Common causes are

HIE

Tuberous sclerosis

Perinatal infections

Metabolic disorder

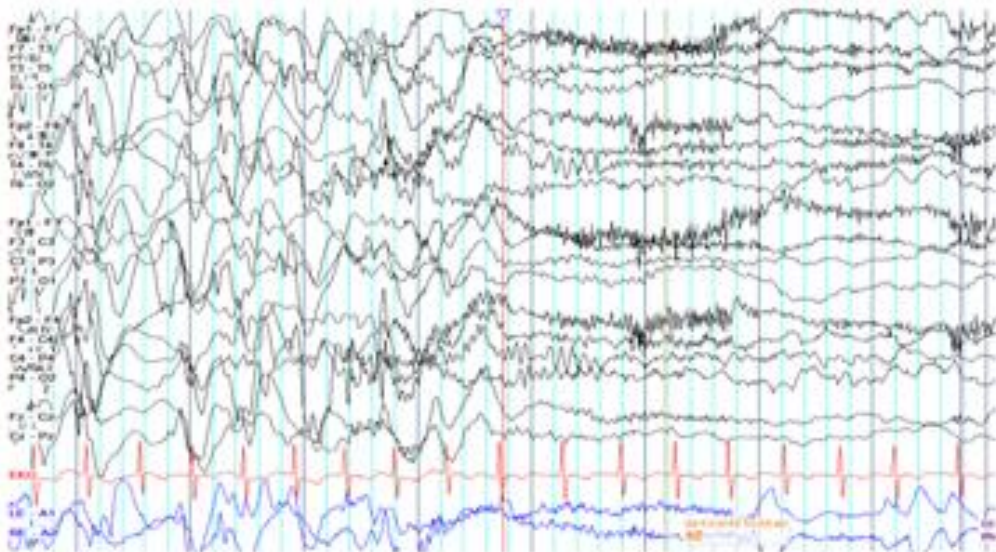
Idiopathic

ARX gene mutation can cause west syndrome in boys with ambiguous genitalia .And the diagnosis is delayed because of mistaking the startle reflex for the colicky pain ATCH and corticosteroids are used on the treatment and vigabatrin is the drug of choice in tuberous sclerosis.

Normal EEG Awake



Infantile Spasms

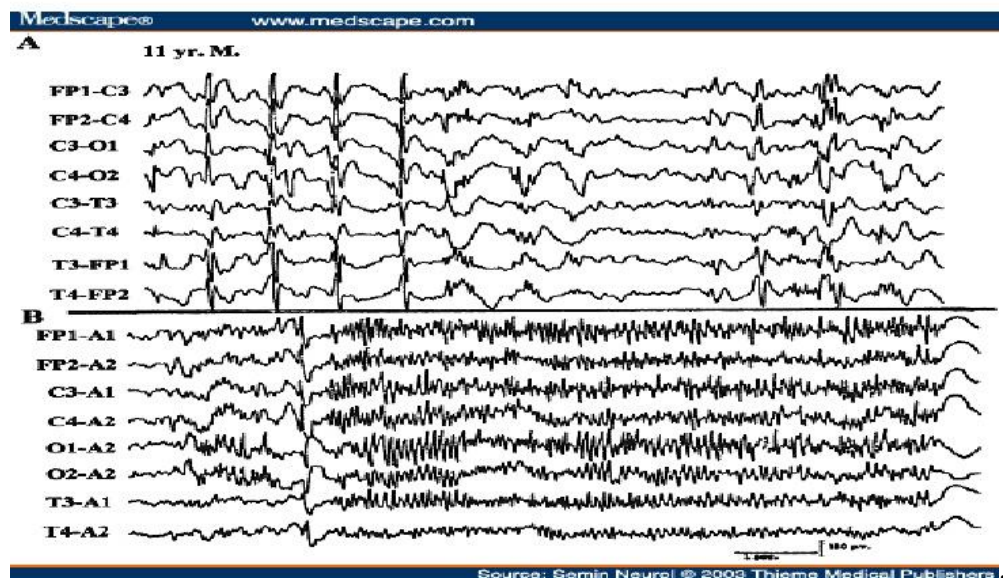


LANNOX- GASTAUT SYNDROME¹

is characterised by multiple seizures types include atypical absences, tonic , astatic, and Myoclonic.

triad similar to west syndrome include development delay, multiple seizures and EEG findings with 1-2Hz spike and slow wave discharge. Slow background and polyspike and wave

EEG of an 11-year-old patient with Lennox-Gastaut syndrome, showing generalized paroxysmal fast activity (B)



Drug of choice is valproic acid, benzodiazepines, ACTH. Prognosis is unsatisfactory.

LANDAU KLEFFNER SYNDROME¹

often confuses with autism since there is loss of language function which is previously acquired normal according to the development. Mean onset of age is 5.5 years. Characterised by auditory agnosia, expressive aphasia, irritability, poor attention span, behavioural problems

ABSENCE SEIZURES

Patient have brief period of abrupt lapse of awareness of surroundings and environments and sudden discontinuation of the activities being performed with staring spell, eye fluttering. Seizure last not more than 30 seconds. Aura, post ictal convulsions are absent. There is no loss of tone and posture. Incontinence of urine stool may occur. Simple automatism may occur in absence seizure like picking the cloth, lip smacking, head falling forward.

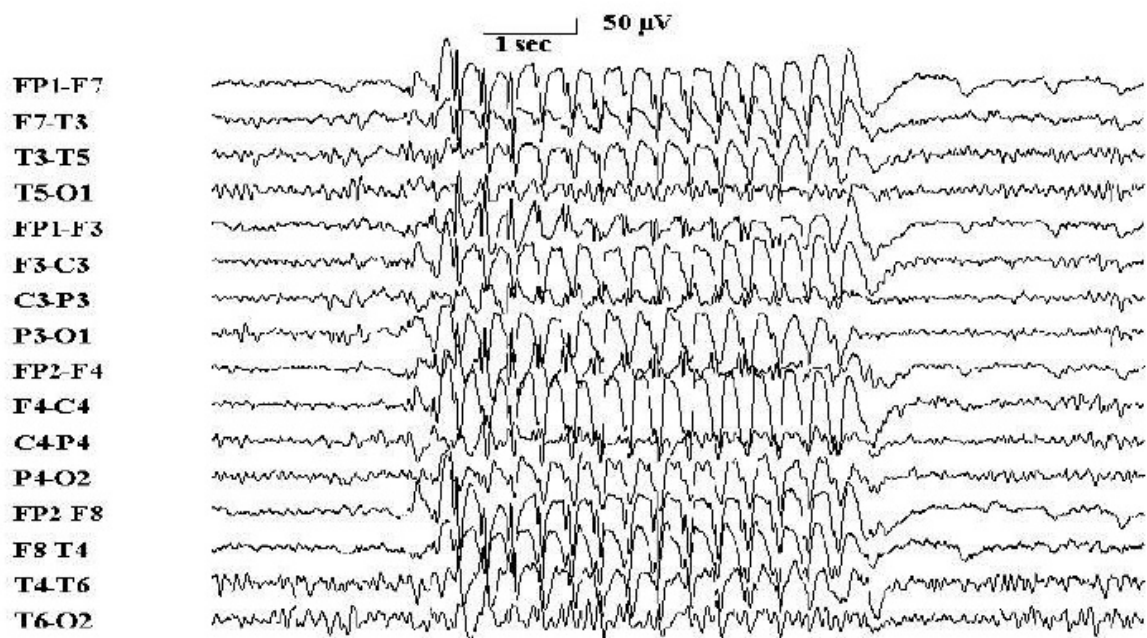
Hyperventilation can precipitate the attacks. Will have characteristic 3per second spike and slow wave pattern

Typical absence seizures are difficult to treat present with myoclonic seizures and change in head and body tone.

It is associated with 1-2 Hz spike and slow wave activity in EEG.

Juvenile absence seizures occurs later age group children similar to typical absence seizures associated with 4-6 Hz spike and slow wave Discharge.

Typical 3-Hz spike and wave discharges seen in absence epilepsy



PARTIAL SEIZURES

Accounts 40% of all seizures on childhood.

Most common causes are inflammatory granulomas, atrophic lesions, vascular insults, birth asphyxia, head trauma, neoplasm.

Simple partial seizures

Begins a focal seizures. Consciousness is not impaired. Symptoms may be motor, sensory. Sometimes visual olfactory, auditory, taste hallucinations.

Often motor seizures include jacksonion march from face to arm to leg.

These seizures are not under voluntary control unlike in tics.

Complex partial seizures

Originates arises from parietal or temporal lobes and may be associated with automatism and loss of consciousness.

These type of seizures last for one to two minutes preceded by aura.

Aura is abnormal feeling such as sense of fear, de ja vu phenomenon, visual hallucination, focal sensation, simple visual experience.

Children less than seven years cannot express or experience aura, but the parents can identify the aura by the children's abnormal behaviour followed by staring look, decreased responsiveness followed by automatism

Automatism is the automatic semi purposeful movements of the extremities and mouth such as chewing, shuffling, walking manipulating sheets.

Secondary generalized seizures

Are simple or complex partial seizures with subsequent generalised seizures.

Nocturnal autosomal dominant frontal lobe epilepsy occurs during night with dystonic posturing. It better responds to carbamazepine

MECHANISMS OF SEIZURES ¹

Epileptic seizures involve excessive firing and synchronisation of neurons. This interrupts the normal working of the part of the brain involved, leading to the clinical symptoms and seismology of the specific type of epilepsy.

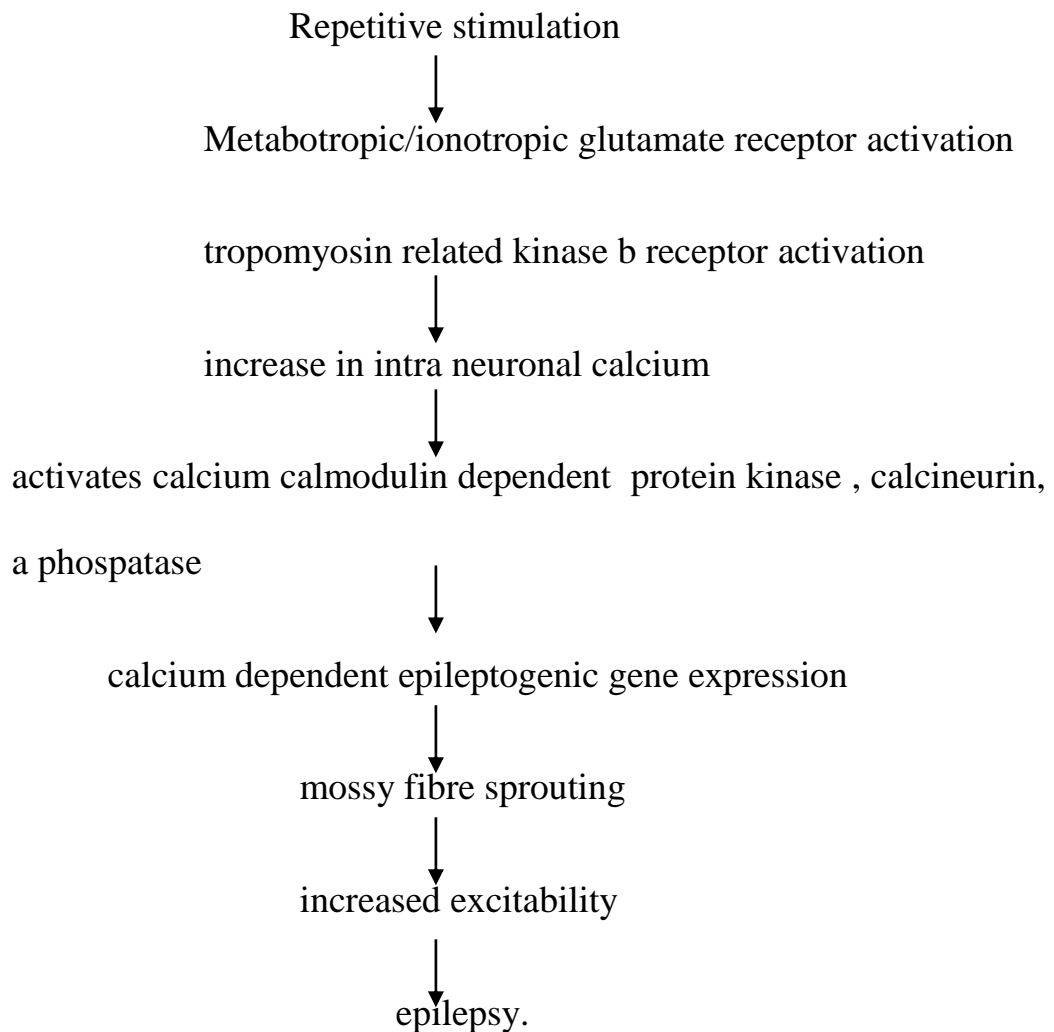
There are four distinct mechanistic processes in path physiology of seizures.

1. Underlying aetiology.
2. Eliptogenesis.
3. Epileptic state of increased excitability
4. Seizure related neuronal injury.

1. Underlying aetiology such as brain tumours, scarring, stroke mutation of specific genes which involves voltage gated channels and ligand gated channels which results in neuronal function disruption and connectivity makes the brain epileptogenesis.

2. Eliptogenesis is the mechanism where the brain becomes epileptic.

Kindling is an animal model where the epilepsy is produced with low intensity repeated electrical stimulation of particular part of a brain for human temporal lobe epilepsy.



3. Epileptic state of increased excitability is irrespective of underlying etiology and mechanism of seizures, it is present in all patients with seizures.

4. Seizure relate neuronal injury is present after prolonged afebrile and febrile status epilepticus. Patient may have hippocampal swelling and hippocampal atrophy with sclerosis. The involved region may have apoptosis and necrosis that particular tissue.

STATUS EPILEPTICUS

Is the continuous recurrent seizure activity lasting for more than thirty minutes without regaining consciousness.

IMPENDING STATUS EPILEPTICUS

Last for 5 to 30 minutes without regaining consciousness.

REFRACTORY STATUS EPILEPTICUS -

Failed to responds at least two medication and last for more than 30 minutes.

Neurological sequelae following status epilepticus depends on aetiology age and duration of seizures. Seizures recur in 25% - 75% of patients. Mortality rate is 10% most deaths are related to patients underlying pathological condition.

In around 50% of the cases, status epilepticus is the first seizures. And about 3% of epileptics experience status epilepticus in their lifetime.

Path physiology of status epilepticus ¹

Excessive and persistent excitation or ineffective recruitment of inhibition. Excitatory neurotransmitters are aspartate, glutamate, acetylcholine and inhibitory neurotransmitter is GABA. Blockade of NMDA receptor by magnesium ion are the important cause of neuronal damage in status epilepticus.

Increased excitability is due to AMPA glutamate receptor desensitization. Intracellular internalization of GABA_A receptor leads to reduction of GABA mediated inhibition.

In status epilepticus, the neuronal injury is due to increased cerebral metabolic rate which results in increase in cerebral blood flow initially. This mechanism is not maintained more than half an hour and leads to inadequate oxygen tension.

Neuronal Apoptosis and necrosis can occur in status epilepticus.

INVESTIGATIONS

All baseline investigations should be done

Complete blood count , renal function test, liver function test, electrolytes , blood sugars should be carried out to exclude meningitis, hypoglycaemia, dyselectrolytemia,

Mainstay of investigations include

1.EEG(Electroencephalogram)

2.computerized tomography

3.Magnetic resonance imaging

EEG

The first recording of the electric field of the human brain was made by the German psychiatrist Hans Berger in 1924 in Jena

Electrical activity is recorded by placing electrodes on the scalp in a specific arrangement. Rythms are recorded and evaluated in terms of rate (hz), amplitude(MV),synchrony , morphology and symmetry.

Rythms are

Beta rythm

-14-20Hz(cycles/sec)- during mental activity/tension recorded in parietal and frontal region.

Alpha rythm-

8-13 Hz(cycles/sec) during awake with closed eyes recorded in occipital region

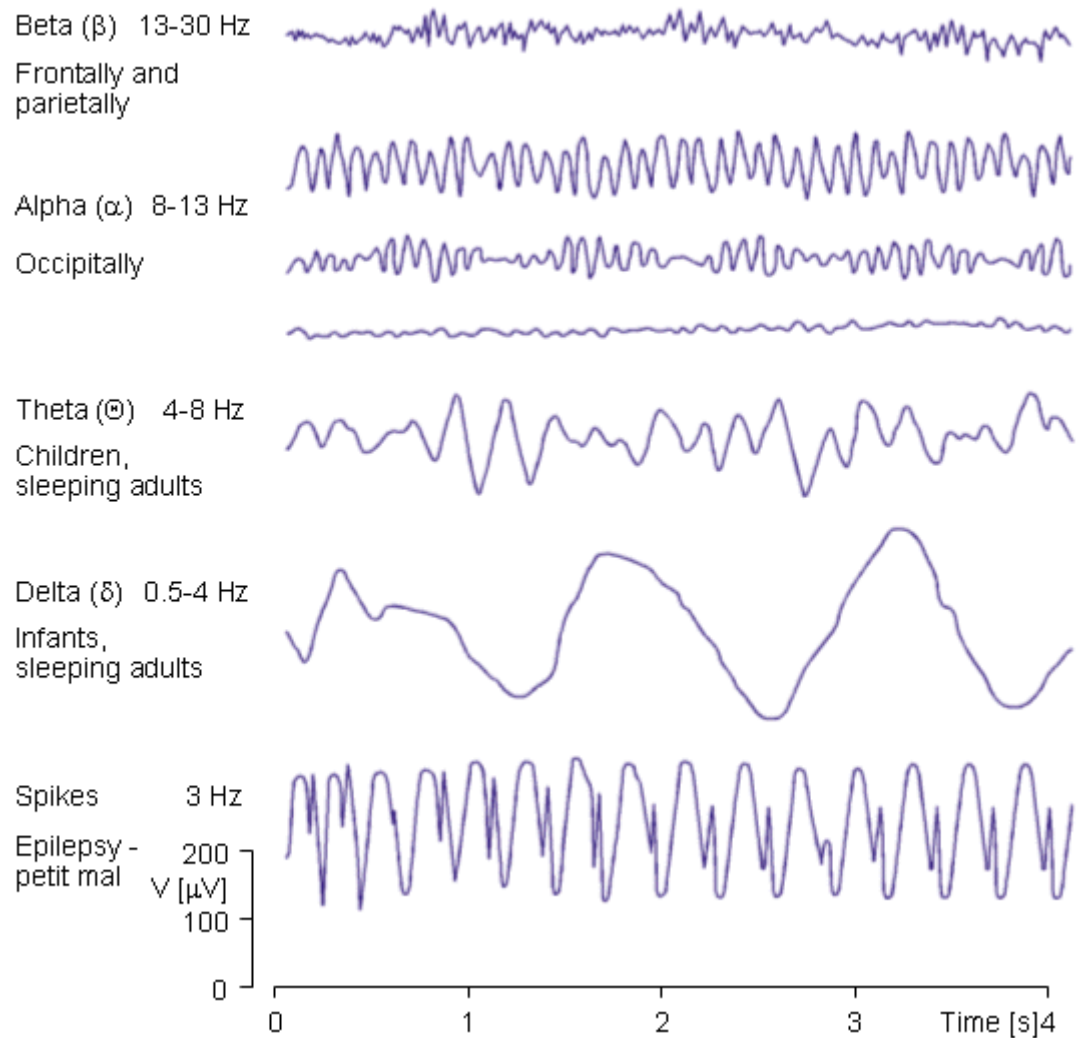
Theta rythm -

4-7 Hz(cycles/sec) occurs in parietal and temporal regions in children normally

Delta rythm -

1-3 Hz(cycles/sec) occurs in deep sleep

NORMAL ELECTROENCEPHALOGRAPH



Normal EEG



Indications for EEG

To classify and to confirm the diagnosis of epilepsy

To identify the possible precipitant to epileptic seizures²

To distinguish between non seizures state and seizures.²

To investigate the cause of cognitive decline ²

To predict the likelihood of recurrence after an initial seizures ²

To monitor the treatment and time of drug withdrawal ²

Interictal EEG usually recorded for 10-60min ²

Most electroencephalogram are recorded between seizures(interictal EEG)²

In diagnosing absence seizure, Myoclonic epilepsy, non convulsive status, epileptic syndrome, sub acute pan encephalitis and herpes encephalitis

EEG is not indicated in febrile convulsions

Common abnormalities are slow abnormal asymmetrical rythms.

These rythms may be localized, lateralised to one side or generalised. Thus it helps us to locate the anatomical locations of the lesions.

Certain spikes and polyspikes any associated with certain epilepsies.

SPIKES

These are the transient discharge that stands out from background. It last less than 70 milliseconds and it is accompanied by slow wave.

When spikes are closely placed then it is termed as polyspikes.

Limitations of EEG

Patients may have normal interictal EEG records with epilepsies

About 2% normal populations may have abnormal EEG records with no clinical consequences. So treatment should be started or discontinued based upon the clinical scenarios and other investigation modalities. Its main usefulness is to predict the recurrences of seizures in the future after discontinuation of the therapy

EEG findings in specific seizures and epilepsies

1. Typical absence attack-A three per second spike and wave discharge
2. Generalised seizures-generalised epileptiform discharges
3. Focal seizures – focal epileptiform discharges
4. Myoclonic epilepsies-brief burst of polyspikes
5. Benign focal epilepsies of childhood-cluster of high amplitude spike wave complexes seen in rolandic areas
6. Infantile spasm-high voltage(>100MV) generalised , chaotic slow waves(hypsarrythmia) and multifocal spikes

7. SSPE(sub acute pan encephalitis)-periodic epileptiform discharges recurring at similar interval
8. Herpes encephalitis-periodic lateralized slow wave or high voltage complexes.
9. Focal slowing-inflammatory granulomas, cerebral infarctions.
10. Encephalitic syndromes-generalised slowing of background activities

NEURO IMAGING

Ultra sonography

It is the investigation of choice in infants and newborn with open anterior fontanel. It is a bedside and easily performed. Ventricle, periventricular tissue, part of cortex are better visualised. posterior fossa and peripheral cortex are poorly visualised.

COMPUTERIZED TOMOGRAPHY

CT brain is readily available and easily taken and cheap in cost. But cannot detect certain epilepsies where MRI brain is the mainstay of investigation.

CT brain mainly able to detect fractures, hemorrhage and space occupying lesions. Calcifications are best evaluated in ct brain.

Advantages of CT are

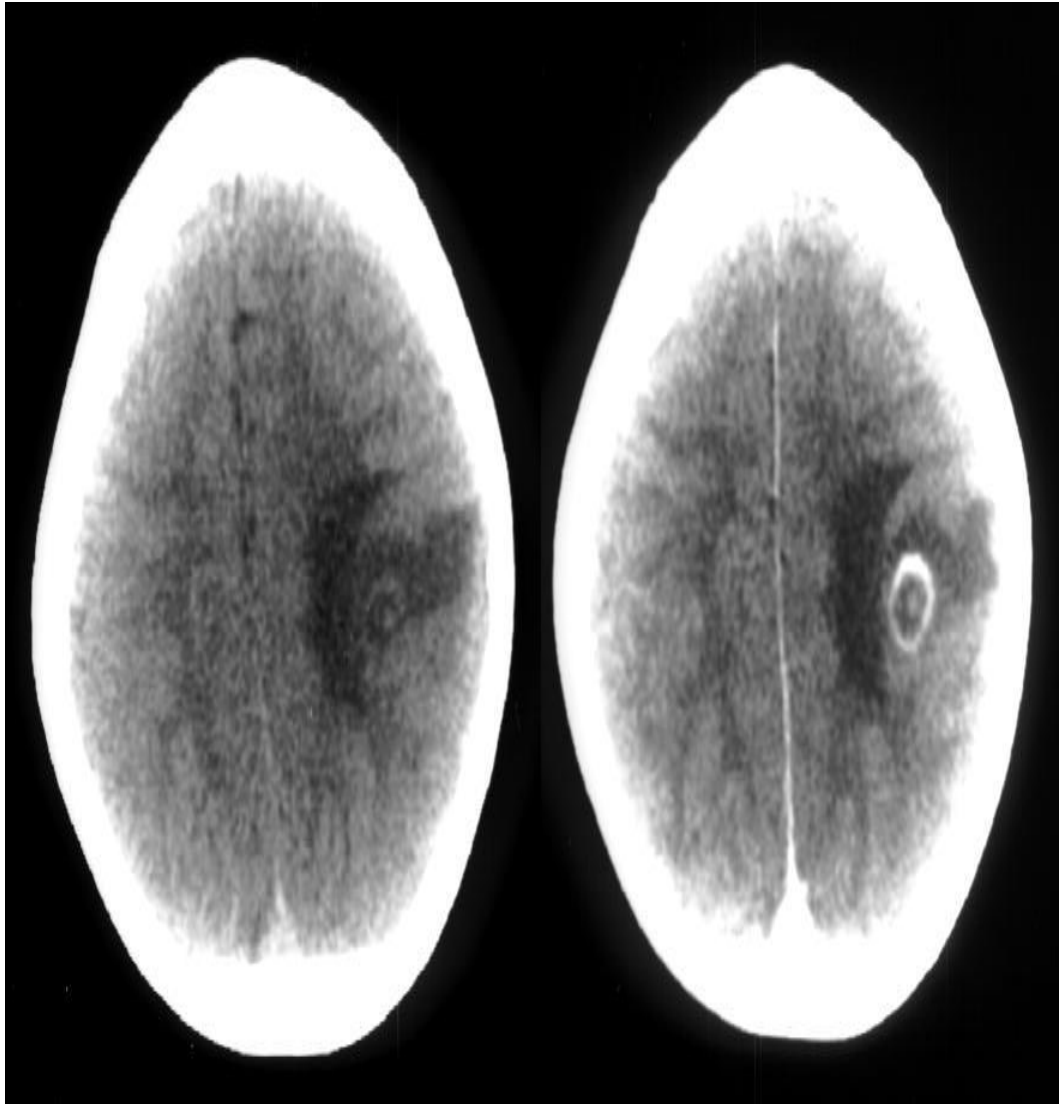
1. Easy availability
2. Fast reliable
3. Better for bone and acute blood lesion, skull study
4. Calcification
5. Less limited by patient factors

Disadvantage of CT are

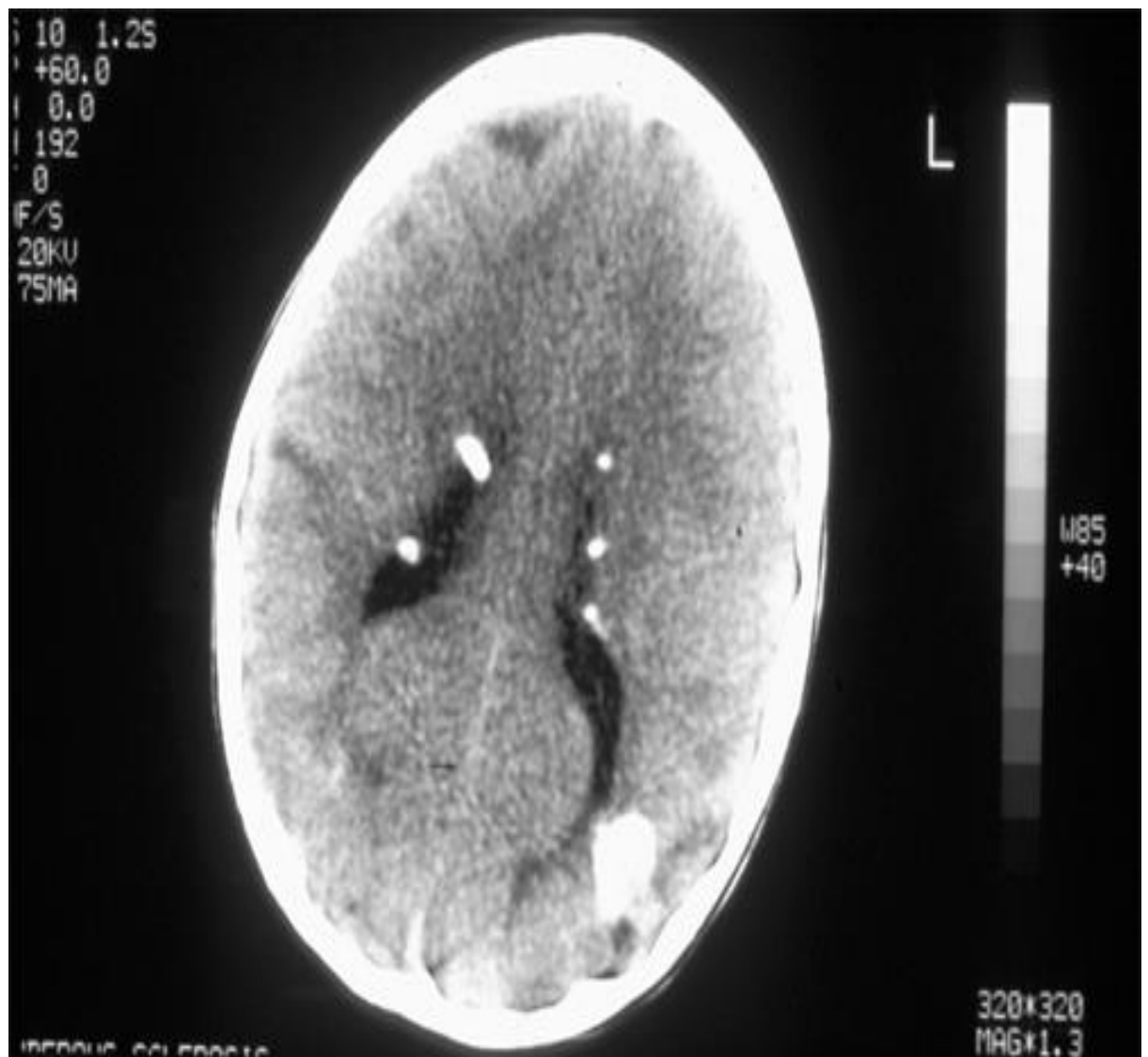
1. High radiations
2. Poor visualisations if posterior structures

Best indicated in

1. HIE
2. Head injury
3. CNS tuberculosis
4. Neurocysticercosis
5. Degenerative brain disorders
6. Hydrocephalus
7. Space occupying lesion



Ring enhancing lesion

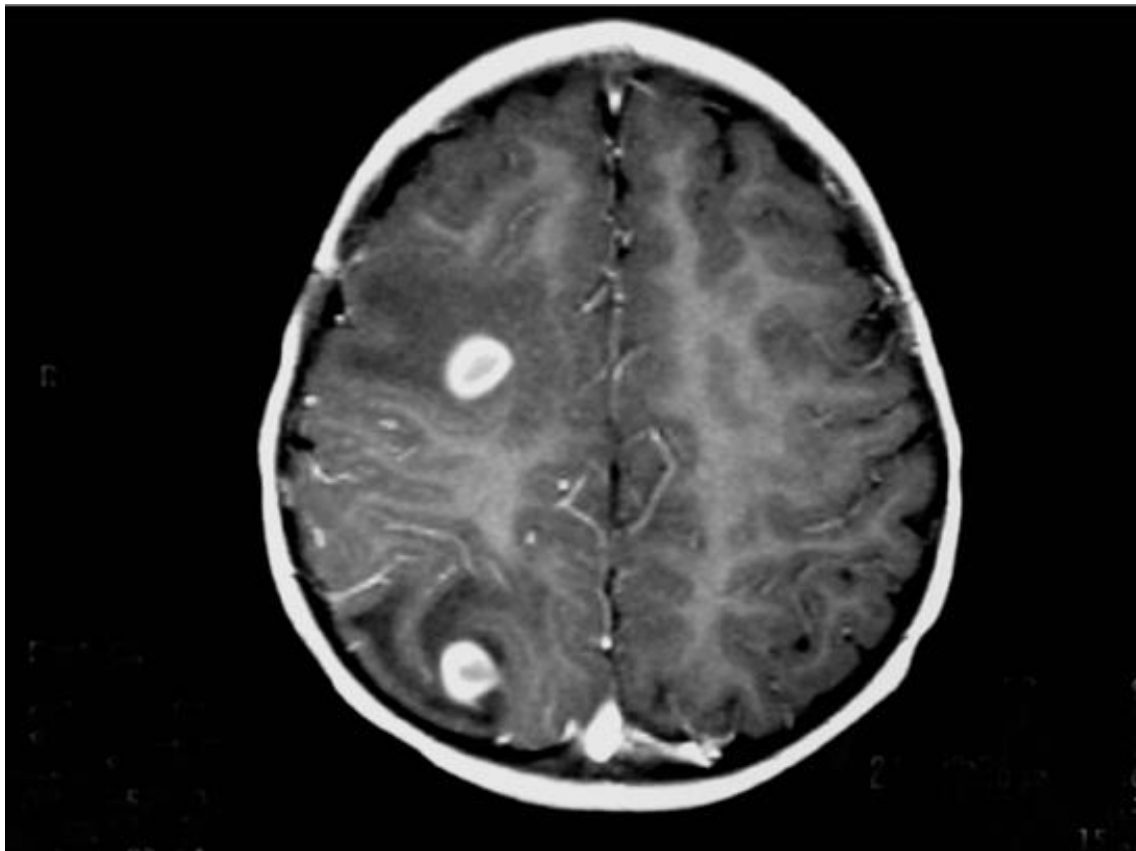


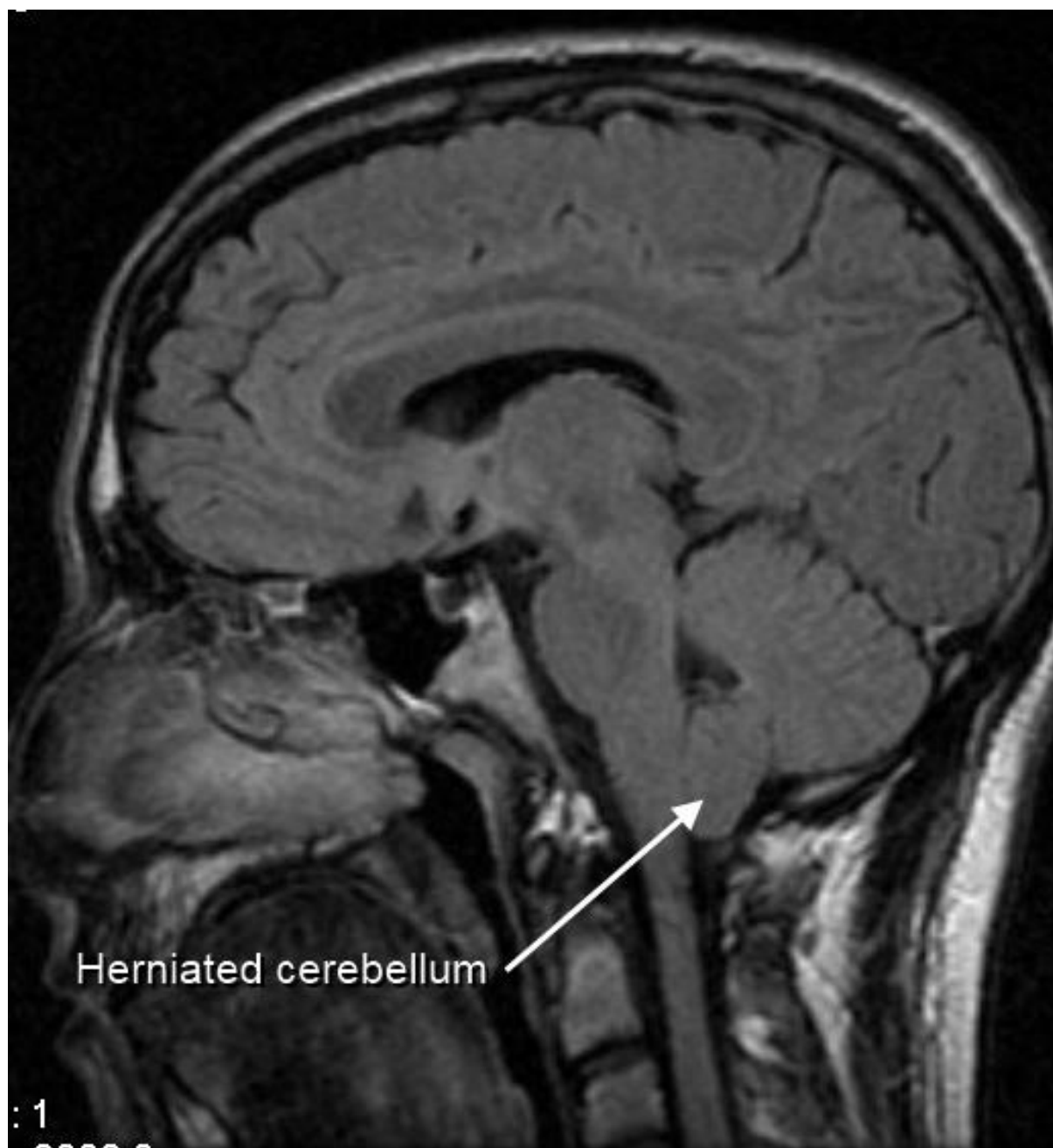
Tuberous sclerosis- cortical tubers

MRI BRAIN

Provides anatomical delineations, grey-white matter distinction , detection of demyelination, congenital vascular anomalies. Midline structures, posterior fossa structures are better visualised. Neurodegenerative disorders are diagnosed early in mri. T1 weighted images are better used to detect brain malformation and maldevelopment. T2 weighted images are used to detect foreign tissues includes tumours and gliotic tissue

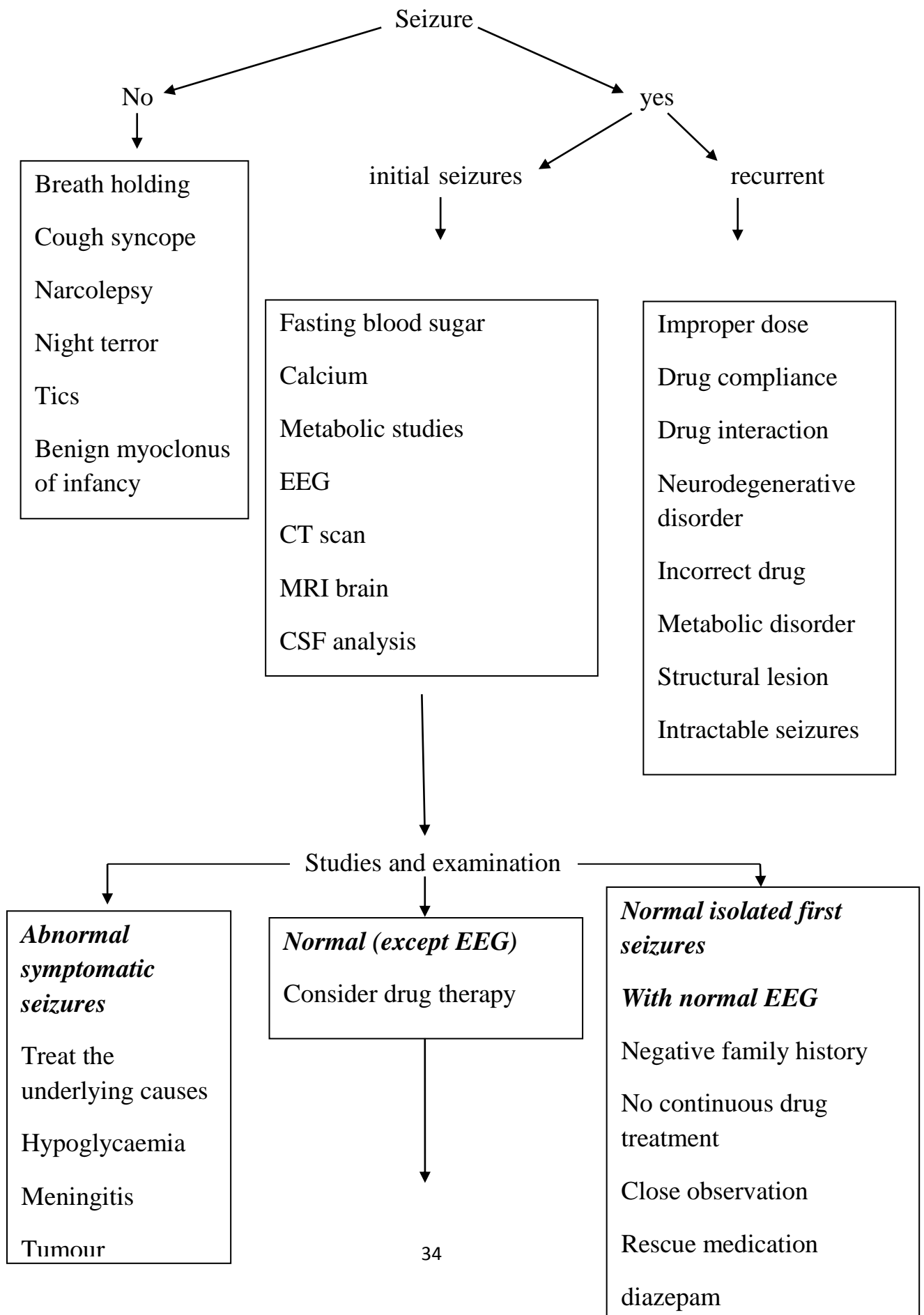
Neurocysticercosis brain mri

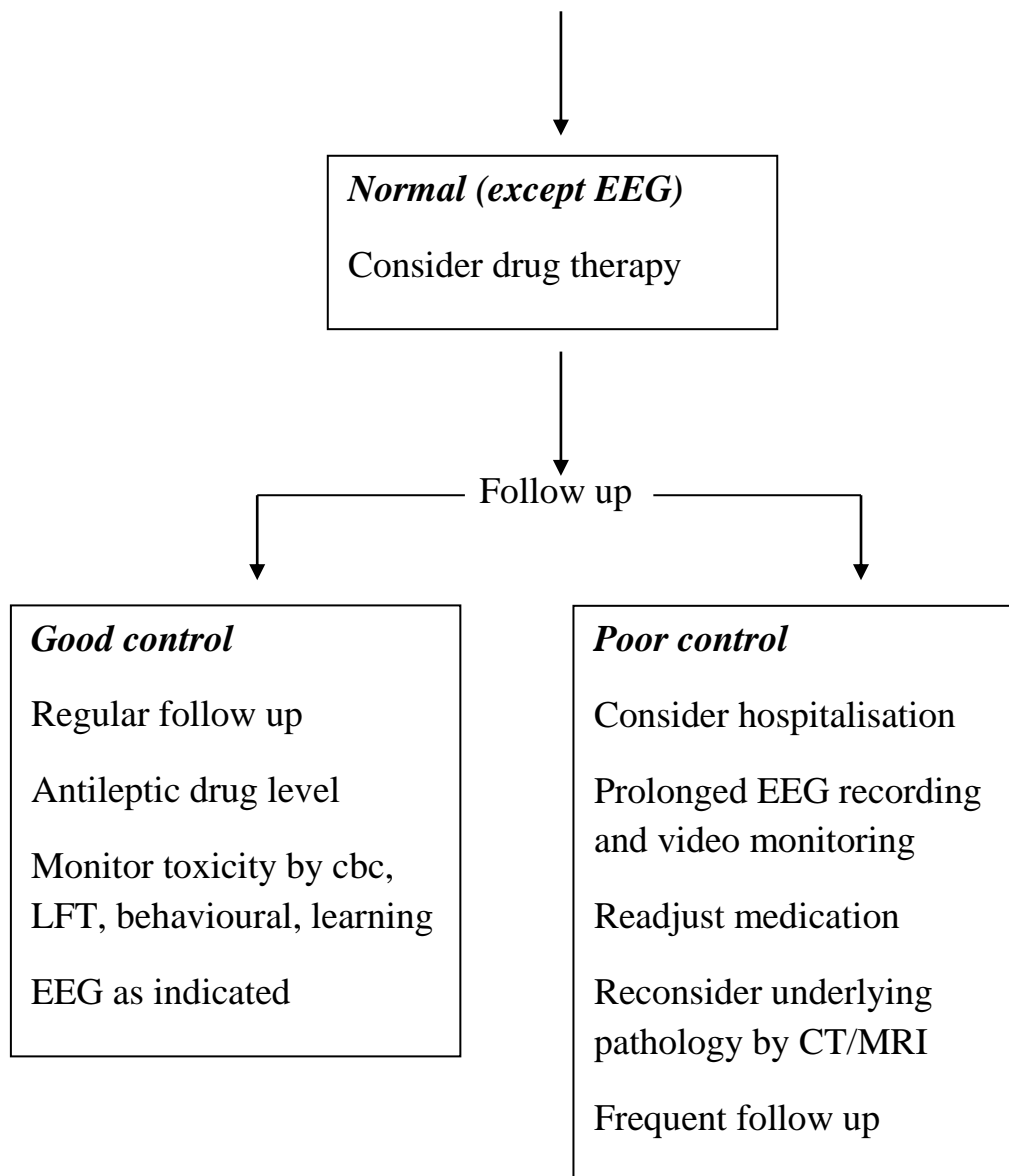




Arnold chiari malformation

TREATMENT OF EPILEPSY¹





Drugs used to treat seizures are

Medications	Dosage mg/kg/day	Usual dosing
Phenobarbitone	5	BD
Phenytoin	5	BD
Valproate	10-40	BD/TDS
Clobazam	0.5	BD/TDS
Diazepam	0.01-0.02 IV	BD/TDS
levetiracetam	20-40	BD/TDS
Lorazepam	0.03	BD/TDS
Carbamezapine	10-20	TDS
Clonazepam	0.01-0.02	BD/TDS

Drug therapy should be initiated based on the epilepsy syndrome and seizure.

First drug of choice for seizures are as follows

1. Focal seizures- carbamazepine.
2. Absence seizures-ethosuximide.
3. Juvenile Myoclonic epilepsy-valproate, lamotrigine.
4. Lennox-gastaut syndrome – valproate,lamotrigine.
5. Infantile spasm- ACTH
6. Status epilepticus-iv diazepam/lorazepam/midazolam

AIM AND OBJECTIVE

To assess the role of Neuroimaging (CT/MRI) and EEG in patient with unprovoked afebrile seizures

And to find out whether there is any correlation between these investigations

Study design - Prospective analysis

Study place - Pediatric department

Tirunelveli medical college and hospital,

Tirunelveli

Study period - From February 2015 To September 2015

Study population- Children presenting with unprovoked afebrile

seizures

Sample size - 50 children

Inclusion criteria - children presenting with unprovoked afebrile

seizures in the age group postneonatal to twelve

years with normal development

epilepsy

Exclusion criteria - Seizures in neonatal period

Acute symptomatic seizures

Febrile seizures

Developmental delay

Post traumatic seizures

Meningitis

Encephalitis

MATERIALS AND METHODS

All children who satisfied the inclusion criteria were included in the study. The data regarding their name, age, sex, type of seizures (according to international classification of epileptic seizures), past history of seizures, drug history, development history, family history are collected in a preformed proforma (Annexure). The examination findings are recorded. EEG recording, CT scan / MRI scan are done. Findings in them are recorded.

REVIEW OF LITERATURE

Ramesh Baheti et al.,⁶ did a study in CT and EEG findings in children with generalised and partial seizures admitted in western rajasthan urban tertiary care teaching hospital.

52 children were included with seizure disorder were study showed 50 % had partial seizures and 50% had generalised seizures.

Abnormal EEG were found in 73% with partial seizure and 76.9% with generalised seizures. CT abnormality were found in 50% in partial seizures and 34.6 % in generalised seizures. It also found that increase abnormalities of EEG, the chance of finding CT abnormality also increases.

Al sulaiman et al²⁷ and doose et al²⁸ done the study of childhood seizures and EEG and found abnormal EEG in 81% with partial seizures and 78% with generalised seizures.

Mcgahan et al reported Abnormal CT in 40% with generalised seizures

Shinnar S et al⁹ did a study in 411 children with first episode of afebrile seizures.

Neuroimaging was performed in 218 patients. 59 MRI brain and 159 Computed tomography were performed. Two patient had neurocysticercosis and two patient had brain tumour. Abnormal neuro imaging were 45(21%). The most common abnormalities were found to be focal encephalomalacia n=16 and cerebral dysgenesis n=11. In both partial seizures and generalized seizures group, the abnormal neuroimaging were found to be similar. Study concluded as neuroimaging should be considered in any child with first episode of afebrile seizures.

Misra et al¹² has done study of neuroimaging finding in seizure disorder among children and done comparison among the CT and MRI brain finding. A total of 96 children were studied. 70 % had abnormal CT imaging. CT abnormalities is found higher incidence in partial seizures(78%) when compared to generalized seizures(65%).ring enhancing lesion 54% is the most common abnormalities found .next is the brain atrophy(12%). Study was concluded with ring enhancing lesion is the most common finding in the CT brain

RESULTS ANALYSIS

Datas were collected and analyzed statistically.

To compare the categorical variables chi square test is used.

To compare the mean values student t test is used.

P value <0.05 is taken as significant.

Table no:1 AGE DISTRIBUTION

AGE GROUP	TOTAL	GENERALISED SEIZURES		PARTIAL SEIZURE	
		n=35		n=15	
		n=50	Mean age	SD	Mean age
		4.82	3.75	5.28	3.58
1M-4 YRS	27	20		7	
5-8 YRS	14	8		6	
9-12 YRS	9	7		2	
TOTAL	50	35		15	

Seizures more commonly occurs in the age group 1 to 4 years.

In our study Generalised seizures are frequently occur in 1 to 4 years 57%.

Partial seizures are frequently occurs in 1 to 4 years 46 %.

In Generalised seizure mean age is 4.82 with standard deviation of 3.75

In Partial seizure, the mean age is 5.28 with standard deviation of 3.58

P value is 0.455 by pearson chi square test which is statistically not significant.

Chart no:1 AGE DISTRIBUTION

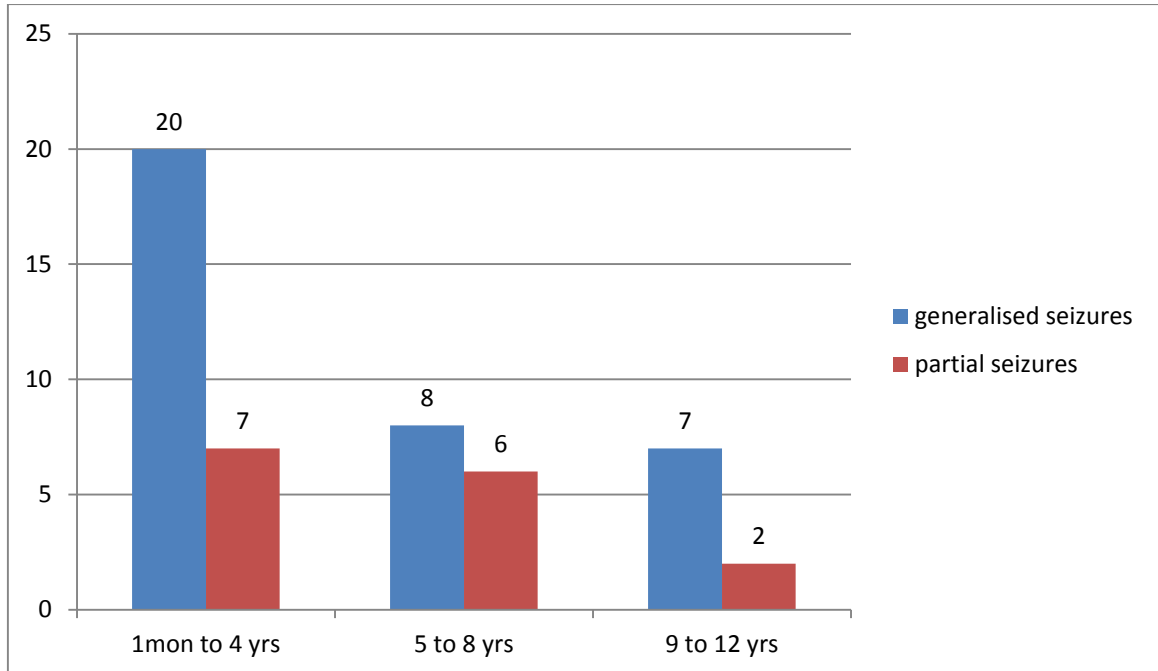


CHART NO : 2 GENERALISED SEIZURES VS PARTIAL SEIZURES

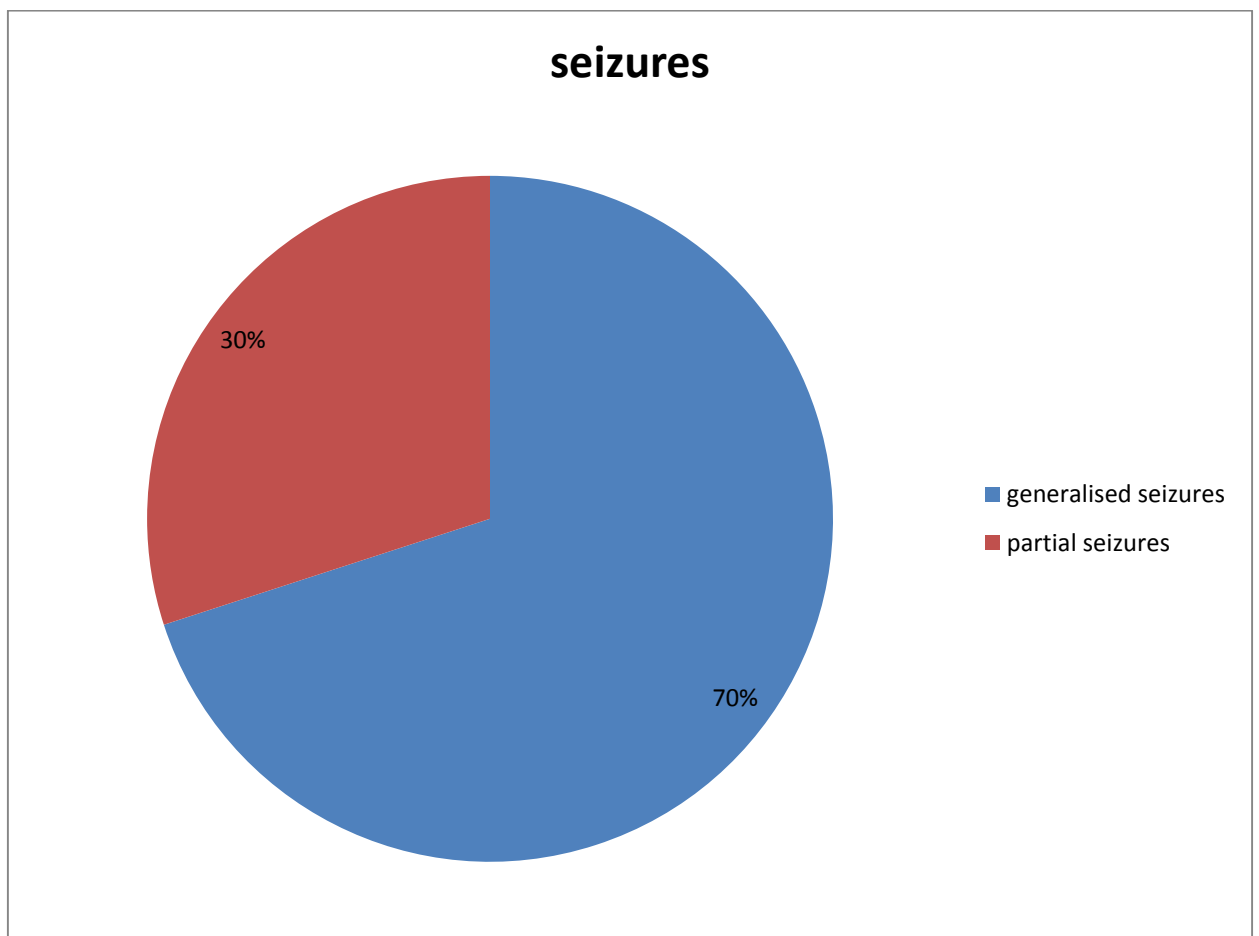


TABLE NO :2 SEX DISTRIBUTION

SEX	GENERALISED SEIZURE	PARTIAL SEIZURE	TOTAL	PERCENTAGE
MALE	24(68.5%)	9(60%)	33	66%
FEMALE	11(31.5%)	6(40%)	17	34%
TOTAL	35	15	50	100%

Generalised seizures more frequently distributed in male childrens-68.5%

Partial seizures are more frequently distributed in male childrens.60%

In generalised seizures male to female ratio is 2.1:1

In partial seizures male to female ratio is 1.5:1

P value is 0.558 by pearson chi square test which is statistically not significant.

CHART NO 3: SEX DISTRIBUTION

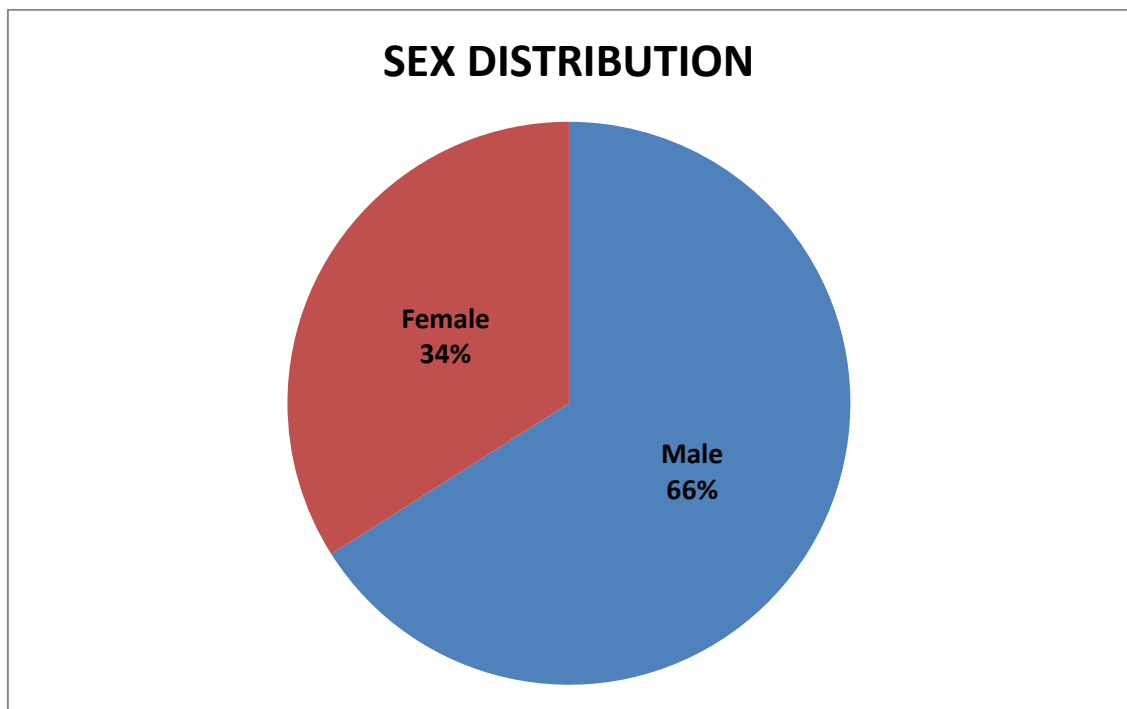


CHART NO 4:SEX DISTRIBUTION

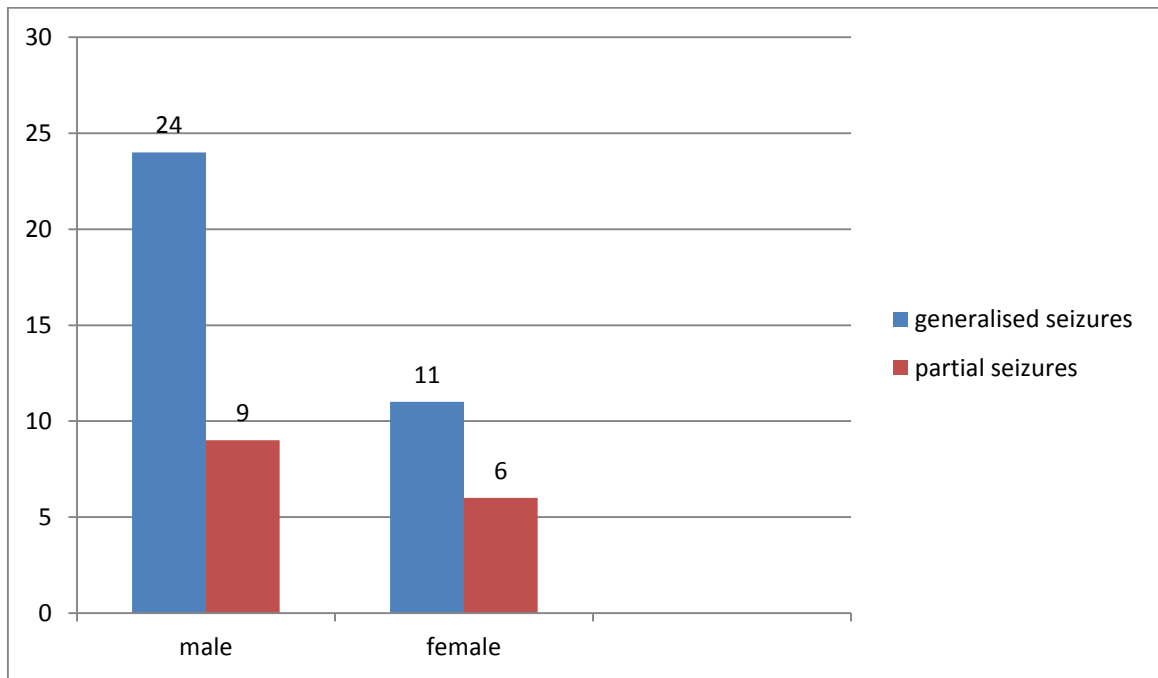


TABLE NO 3: FAMILY HISTORY OF SEIZURES

TYPE	PRESENT	ABSENT	TOTAL
GENERALISED SEIZURE	7	28	35
PARTIAL SEIZURE	3	12	15
TOTAL	10(20%)	40(80%)	50

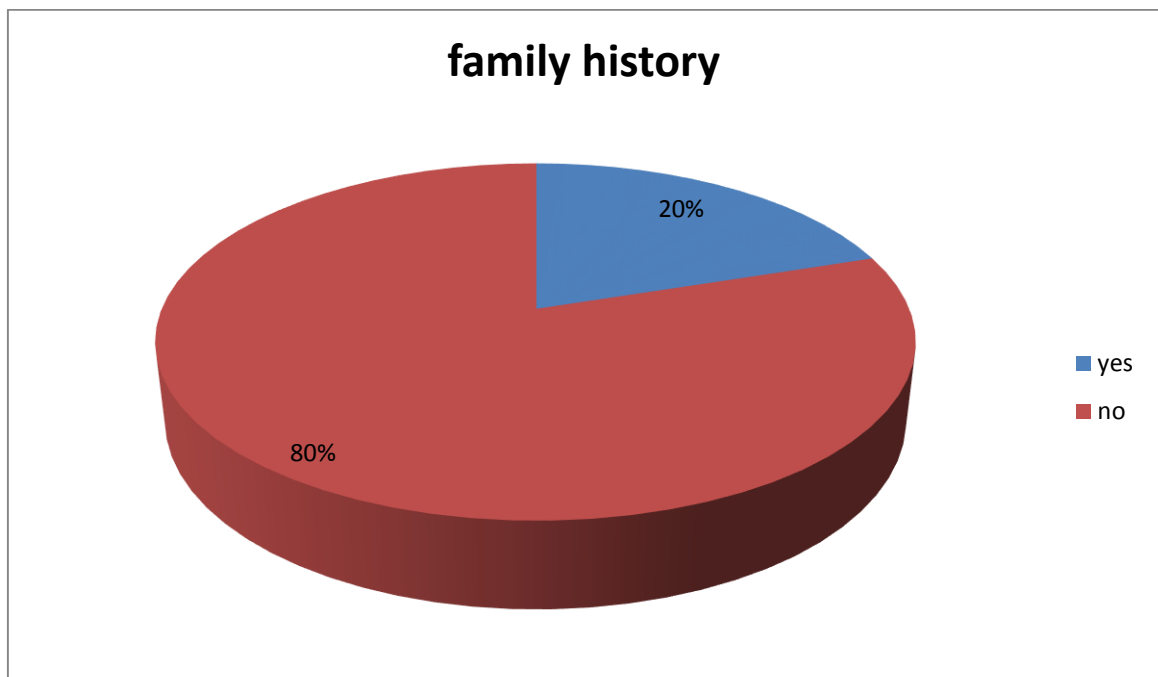
20% of the patient has family history of seizures.

20 % of the children among generalised seizures has family history

20 % of the children among partial seizures has family history of seizures.

P value is 1 by Fisher's exact test. There is no significant difference in the family history in partial and generalised seizures.

CHART NO 5: SEX DISTRIBUTION



**CHART NO 6: FAMILY HISTORY OF SEIZURES IN
GENERALISED & PARTIAL SEIZURES**

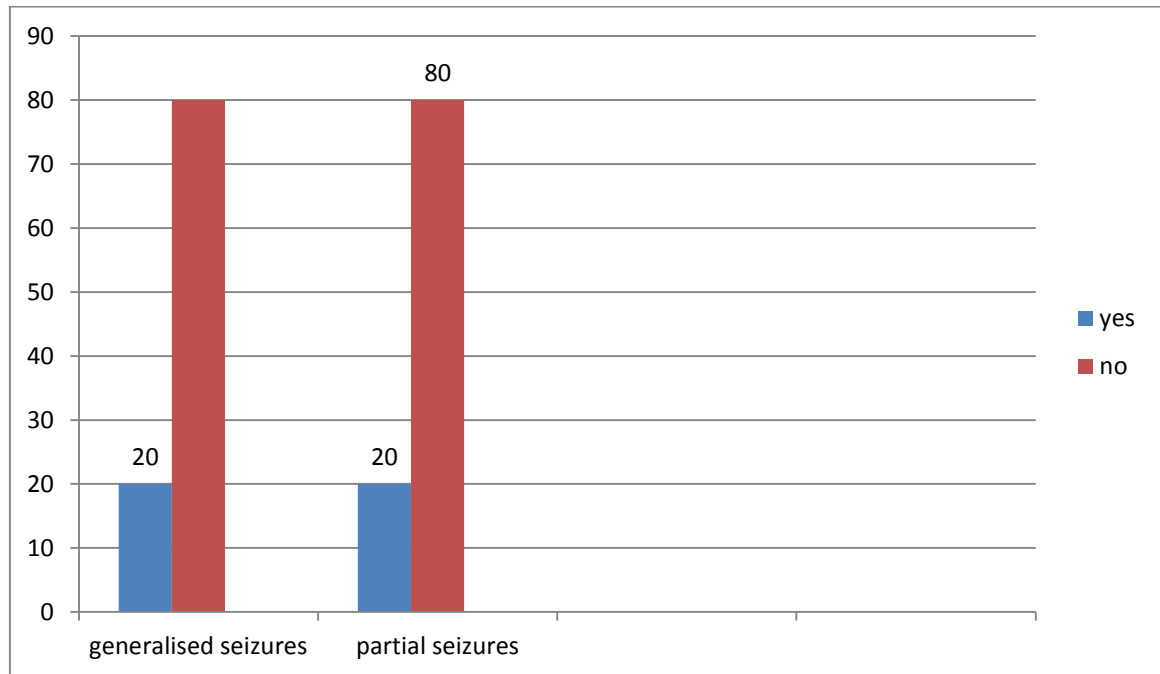


TABLE NO 4: EEG FINDINGS

SEIZURE TYPE	ABNORMAL	NORMAL	TOTAL
GENERALISED	30(85.7%)	5(14.3%)	35
PARTIAL	11(73.3%)	4(26.7%)	15
TOTAL	41(82%)	9(18%)	50(100%)

Among the 50 children admitted with seizures in the study 82% had EEG abnormality.

85.7% among the generalised seizures had EEG abnormality

73.3% among the partial seizures had EEG abnormality

P value-0.423- Fisher's exact test

There is no statistically significant difference in the incidence of the EEG abnormality among the partial and generalised seizures.

CHART NO 7: EEG FINDINGS IN GENERALISED & PARTIAL SEIZURES

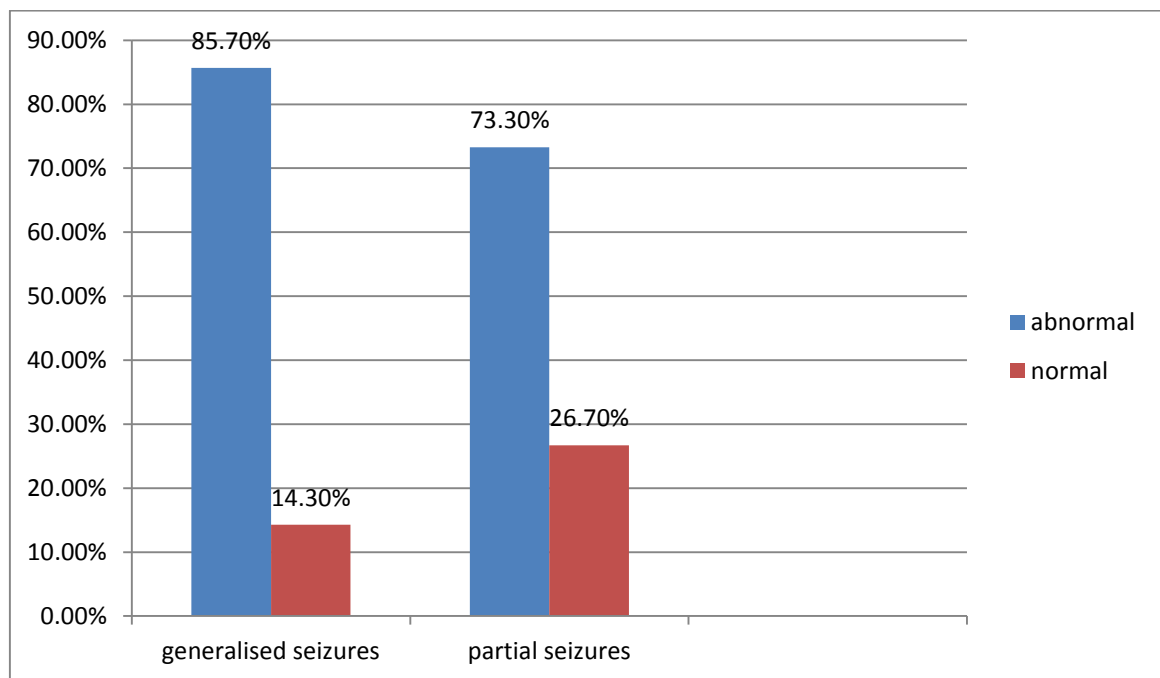
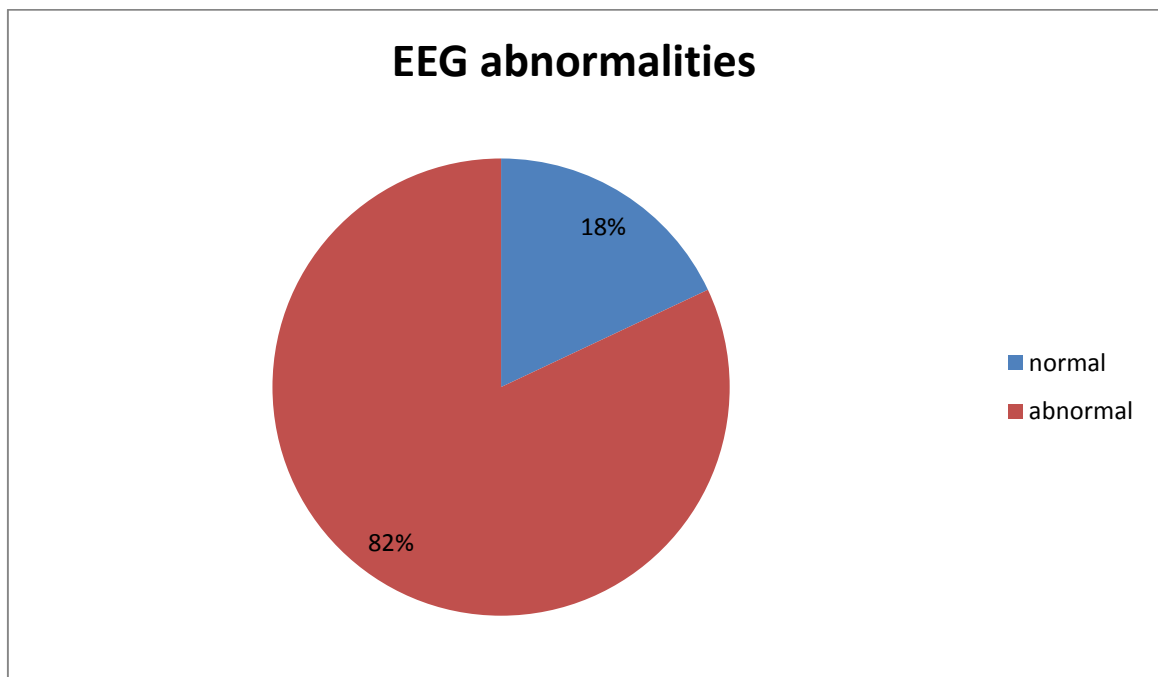


CHART NO 8: EEG ABNORMALITY IN SEIZURES



**TABLE NO 5: GENERALISED AND FOCAL EEG CHANGES IN
GENERALISED & PARTIAL SEIZURES**

SEIZURE	GENERALISED EEG ACTIVITY	FOCAL EEG CHANGES	TOTAL EEG changes	Normal EEG
GENERALISED	23	7	30	5
PARTIAL	2	9	11	4
TOTAL	25(50%)	16(32%)	41	9(18%)

Among the 50 children included in the study 32% had focal EEG changes.

50% had generalised EEG changes and 18% had normal EEG findings.

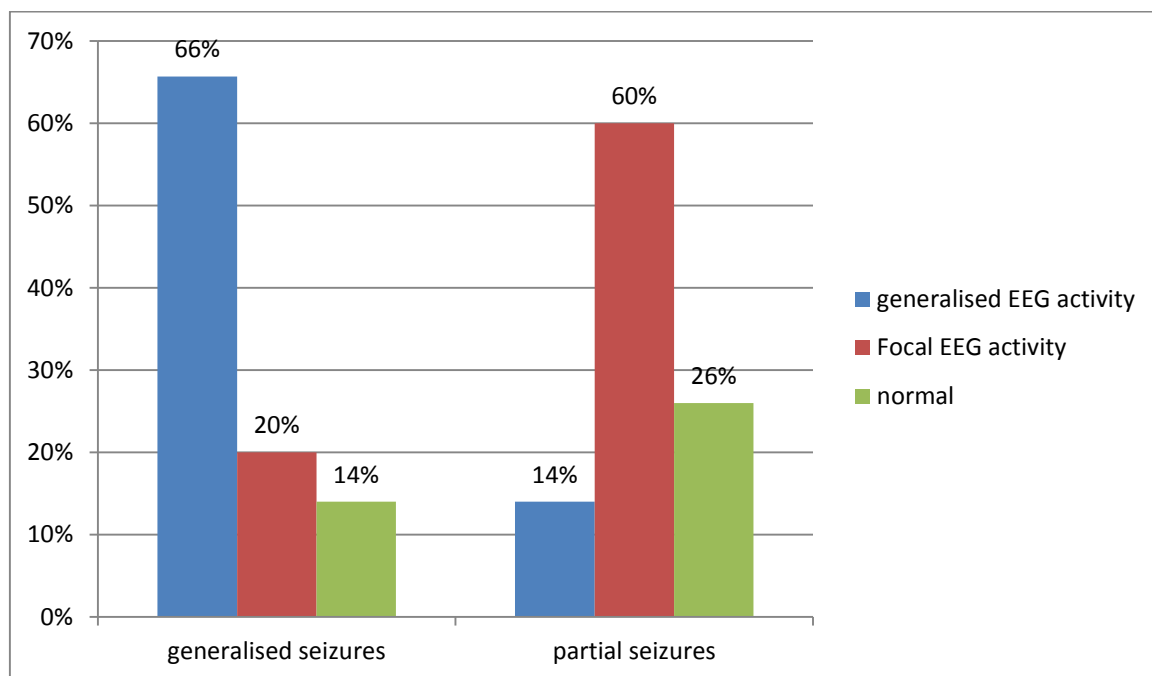
Children with generalised seizures had 65.7% of generalised EEG activity and 20 % had focal EEG activity and 14.3% had normal EEG

Children with partial seizures had 60 % focal EEG changes and 13.3% generalised EEG changes and 26.6% had normal EEG changes.

There is significant higher incidence of generalised EEG changes in generalised seizures when compared to partial seizures group.

P value-0.012 by Fisher's exact test

**CHART NO 9: EEG ABNORMALITY IN GENERALISED &
PARTIAL SEIZURES**



**TABLE NO 6:NEUROIMAGING IN GENERALISED & PARTIAL
SEIZURES (CT/MRI)**

Seizures	Normal neuroimaging	Abnormal Neuroimaging	Total
Generalised seizures	32(92%)	3(8%)	35
Partial seizures	9(60%)	6(40%)	15
Total	41(82%)	9(18%)	50(100%)

+

Among the 50 patient, 44 patient took CT brain and 9 patient took MRI.

Three patient took both CT brain and MRI brain

40 % of the patient with partial seizures have Neuroimaging abnormality.

Only 8% of the patient with generalised seizure have Neuroimaging abnormality.

P value is 0.00803. The result is statistically significant.

There is high correlation between partial seizures and abnormal Neuroimaging.

CHART NO 10: NEUROIMAGING IN GENERALISED & PARTIAL SEIZURES (CT/MRI)

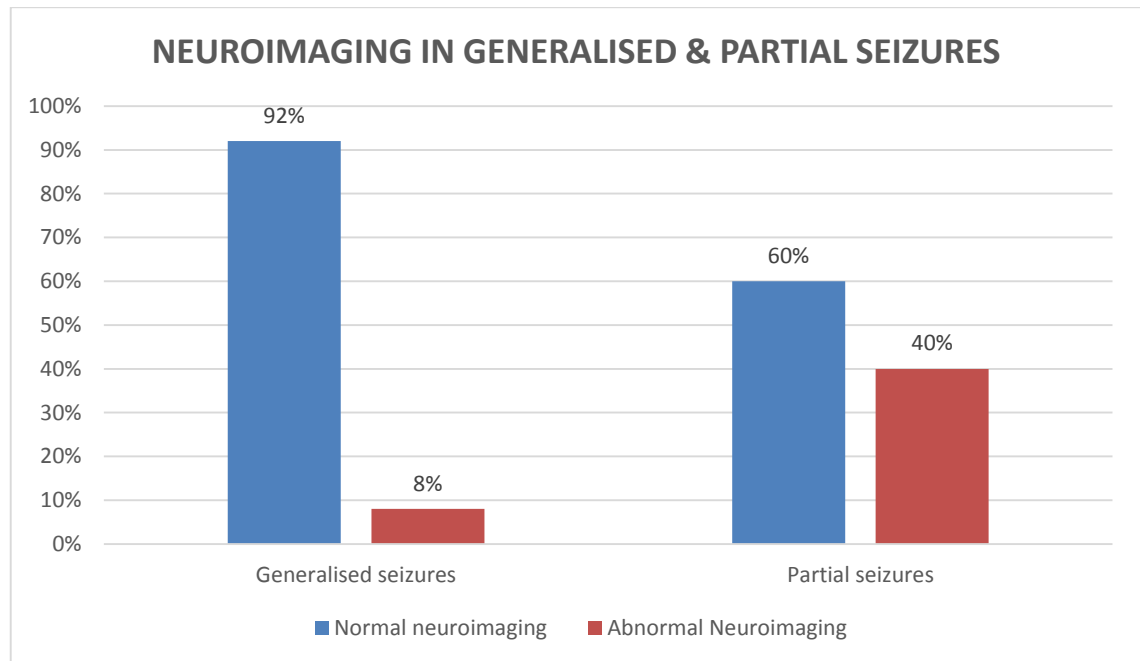


TABLE NO 7:CT FINDINGS IN GENERALISED AND PARTIAL SEIZURES

SEIZURE TYPES	NORMAL	ABNORMAL	TOTAL
GENERALISED	28	2	30
PARTIAL	8	6	14
TOTAL	36	8	44

Among the 50 children included in the study 44 patients took ct brain and 8 patient had abnormal Neuroimaging.

40% among the partial seizures had abnormal CT brain

53 % among partial seizures had normal CT brain

6 % among the generalised seizures had abnormal CT brain

80 % among the generalised seizures had normal CT brain

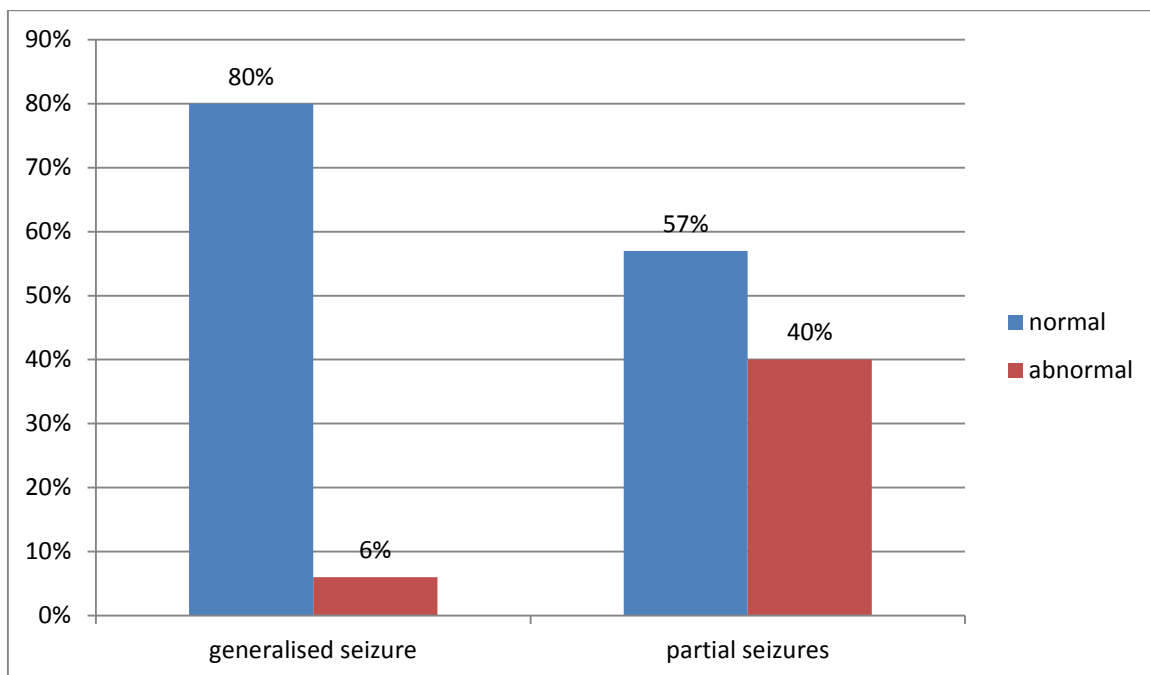
There is significant higher incidence of abnormal CT imaging among the partial seizures

P value is 0.008 by Fisher's exact test

CT FINDINGS

1. Ring enhancing lesion	-	2
2. Atrophic changes	-	1
3. Bilateral gliosis of parietal lobe	-	1
4. Subdural hemorrhage	-	1
5. Dilated 3 rd ventricle	-	1
6. Cortical tubers	-	1
7. White matter hypodensity	-	1

**CHART NO 11:CT FINDINGS IN GENERALISED &N PARTIAL
SEIZURES**



**TABLE NO 8:MRI FINDING IN GENERALISED AND & PARTIAL
SEIZURES**

SEIZURE TYPE	NORMAL	ABNORMAL	TOTAL
GENERALISED	4 (45%)	1 (11%)	5
PARTIAL	1 (11%)	3 (33%)	4
TOTAL	5	4	9

Among the 50 children with seizures 9 patients took MRI brain.

Only 20 % had abnormal MRI imaging among generalised seizures whom MRI was taken

75% % had abnormal MRI imaging among partial seizures whom MRI was taken

There is no significant difference among abnormal MRI neuroimaging between partial and generalised seizures.

P value is 0.206 by Fisher's t test.

MRI BRAIN

1. Ring enhancing lesion	-	1
2. Arnold chiari malformation	-	1
3. Cortical tubers	-	1
4. Acute subdural bleed	-	1

**CHART NO 12:MRI BRAIN ABNORMALITY IN GENERALISED
AND PARTIAL SEIZURES**

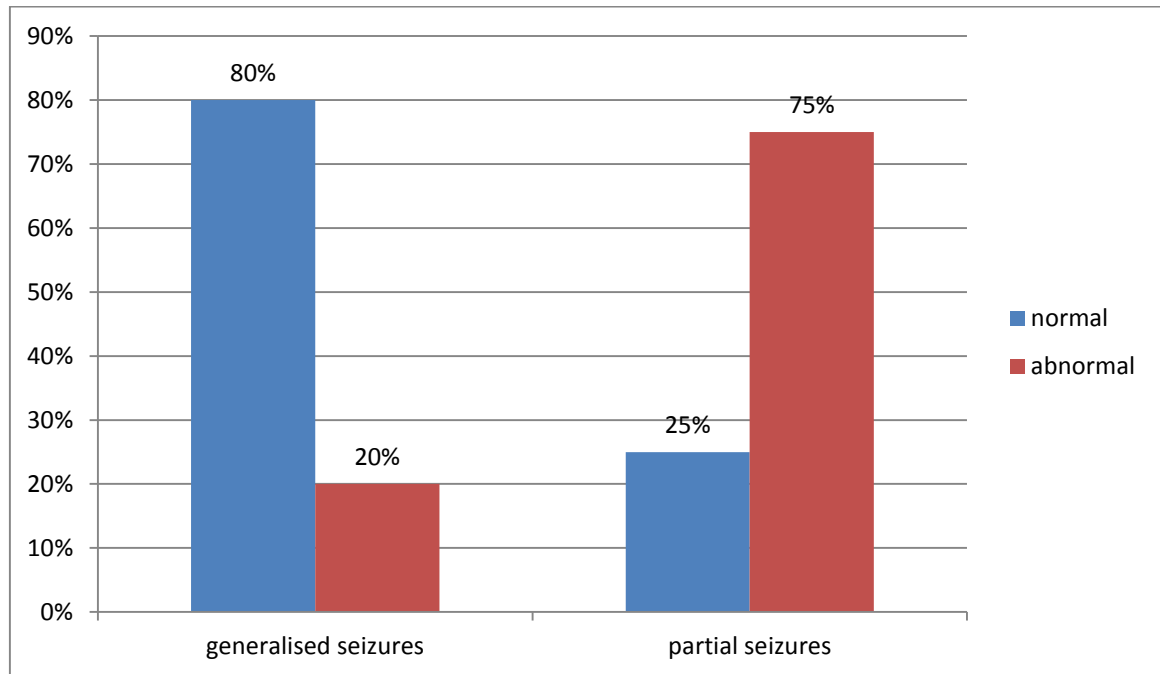


TABLE NO 9:VARIOUS NEUROIMAGING FINDINGS

Neuroimaging	Partial seizures	Generalised seizures
Normal finding	9	32
Abnormal finding	6	3
Ring enhancing lesion	2	
Atrophic changes		1
bilateral gliosis of parietal lobe	1	
subdural hemorrhage	1	
dilated 3 rd ventricle		1
cortical tubers	1	
white matter hypodensity	1	
arnold chiari malformation		1

**TABLE NO 10:NEUROIMAGING ABNORMALITIES IN
PATIENTS WITH NORMAL AND ABNORMAL EEG**

Type of seizures	Normal EEG		Generalised EEG		Focal EEG	
	Normal Neuro Imaging	Abnormal Neuro Imaging	Normal Neuro Imaging	Abnormal Neuro Imaging	Normal Neuro Imaging	Abnormal Neuro Imaging
Generalised seizures	5(100%)	-	22(95%)	1(5%)	7(87%)	1(13%)
Partial seizures	3(75%)	1(25%)	2(100%)	-	4(50%)	4(50%)
Total	9		25		16	

Patients with generalized seizures with focal EEG changes, 87% had normal neuroimaging and 13% had abnormal neuroimaging.

Patients with partial seizures with focal EEG changes, 50 % had normal neuroimaging and 50 % had abnormal neuroimaging

**TABLE NO 11:NEUROIMAGING ABNORMALITIES IN
PATIENTS WITH NORMAL AND ABNORMAL EEG**

	Normal EEG		Abnormal EEG	
	Generalised seizures	Partial seizures	Generalised seizures	Partial seizures
Normal Neuroimaging	5(100%)	3(75%)	27(90%)	6(54%)
Abnormal neuroimaging	-	1(25%)	3(10%)	5(46%)
Total	5	4	30	11

Among children with generalised Seizures with abnormal EEG, only 10 % had abnormal neuroimaging finding

Among children with partial seizures with abnormal EEG, 46% had abnormal neuroimaging.

P value is 0.043. p value is statistically significant. There is high correlation between patient with abnormal EEG having abnormal Neuroimaging.

DICSUSSION

The study is conducted to assess the role of neuroimaging and Electroencephalogram among the patients admitted with generalised and partial seizures. And to find out any correlation between the two investigations

1. The study was conducted among 50 children admitted with seizures and found

35 had generalised seizures (70%)

15 had partial seizures (30%)

Majority of the patients had generalised seizures

2. Age distribution

In Generalised seizure mean age is 4.82 with standard deviation of 3.75

In Partial seizure, the mean age is 5.28 with standard deviation of 3.58

This observation is comparable to the study of Ramesh Baheti et al

3. Sex distribution

Male to female ratio in our study is 1.9:1

In generalised seizures male to female ratio is 2.1:1

In partial seizures male to female ratio is 1.5:1

In our study males are more affected in both generalised and partial seizures

This result is comparable to the study of ramesh baheti et al

3. Family history

Family history is present in 20% of all seizures

There is no statistically significant difference in the presence of family history between generalised and partial seizures in our study

Redda Tekle-Haimanot et al observed 22% family history.

4. EEG finding

EEG were abnormal in 82% of all the children with seizures included in the study.

85.7% among the generalised seizures had EEG abnormality. 73.3% among the partial seizures had EEG abnormality.

Ramesh Baheti et al observed generalised seizure had 76.9 % abnormal EEG activity and partial seizures had 73% abnormal EEG activity

Kurupath radhakrishnan et al observed EEG abnormality in 83.6% of childhood seizure in his study and generalised in 74% of childrens.

Luiz Eduardo betting et al observed EEG abnormality in 33 % of generalised seizures.

6. Computed tomography

In our study 40% among the partial seizures had abnormal CT brain. 6 % among the generalised seizures had abnormal CT brain.

Overall 18.1% had abnormal CT finding among CT taken in our study. And 81.9% had normal CT finding in children in our study.

In our study ring enhancing lesion is the most common finding in the partial seizure group.

Ramesh Baheti et al, abnormal CT brain finding were observed in 50% of partial seizures and 35 % of generalised seizures.

Hussain Jagar et al observed 68% of children with partial seizures had abnormal CT imaging

Misra et al observed 75% of study group had CT abnormality and neurocysticercosis was the most common abnormality.

In our study there is significant statistical difference observed in CT imaging abnormality in partial seizures than generalised seizures

CT imaging abnormality is more common in partial seizures.

This is comparable to the study done by Annie T Berg et al

6. MRI imaging

In our study, Among the 50 children with seizures 9 patients took MRI brain.

Only 20 % had abnormal mri imaging among generalised seizures whom mri taken

Only 75% % had abnormal mri imaging among partial seizures whom mri taken

Mri imaging is more common in partial seizures.

There is no significant difference among abnormal mri neuroimaging between partial and generalised seizures.

In our study

Among children with generalised Seizures with abnormal EEG, only 10 % had abnormal neuroimaging finding

Among children with partial seizures with abnormal EEG, only 46% had abnormal neuroimaging

Patients with generalized seizures with focal EEG changes, 87% had normal neuroimaging and 13% had abnormal neuroimaging.

Patients with partial seizures with focal EEG changes, 50 % had normal neuroimaging and 50 % had abnormal neuroimaging

.

SUMMARY AND CONCLUSION

82% the children with seizures had abnormal Electroencephalogram.

73.1% had shared by generalised seizures and 26.9 % shared by partial seizures.

Higher incidence of neuroimaging abnormality is seen partial seizures with abnormal Electroencephalogram

There is higher correlation between neuroimaging abnormality and focal Electroencephalogram changes in partial seizures when compared to generalised seizures.

Electroencephalogram and Neuroimaging are mandatory in evaluating Children with afebrile seizures.

PROFORMA

- Name
- Age
- Sex
- Date
- Seizure
- age of onset
- episode
- type
- family history
- Birth history
- Developmental history
- Drug history
- Seizure classification
- CT brain
- MRI brain
- EEG

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Abbreviation

Master chart

ABBREVIATION

ACTH	ADRENOCORTICOTROPIC HORMONE
CNS	CENTRAL NERVOUS SYSTEM
CSF	CEREBROSPINAL FLUID
CT	COMPUTED TOMOGRAPHY
EEG	ELECTROENCEPHALOGRAPH
GABA	GAMMA AMINOBUTYRIC ACID
GTCS	GENERALISED TONIC CLNIC SEIZURES
HIE	HYPOXIC ISCHEMIC ENCEPHALOPATHY
MRI	MAGNETIC RESONANCE IMAGING
NMDA	N-METHYL D-ASPARTATE RECEPTOR

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Name	Age	Sex	Ip	family history	Diagnosis	Clinically	Development	Mri	Ct	EEG
krithika	10months	F	42546	no	seizure disorder	generalised seizures	N		n	bilateral epileptiform activity
Karthick	11yrs	M	35787	no	Seizure disorder	generalised seizures	N		N	bilateral epileptiform activity
Logesh	11yrs	M	44513	no	seizure disorder	generalised seizures	N		N	bilateral epileptiform activity
logesh	11yrs	M	32487	no	seizure disorder	generalised seizures	N		n	focal spike and wave discharge
Methuselvam	11yrs	M	3030	no	seizure disorder	generalised seizures	N		N	N
sakthiram	11yrs	M	42321	yes	seizure disorder	generalised seizures	N		n	bilateral epileptiform activity
Sujin	11yrs	M	41234	no	Tuberculoma	partial seizures	N		ring enhancing lesion	focal spike and wave discharge
sumathy	11yrs	F	6128	no	seizure disorder	partial seizures	N		N	r frontal central sharp wave
sivapriya	12yrs	F	29314	no	absence seizure	generalised seizures	N		n	3hz spike and wave discharge
Praveen	1yr	M	44062	no	seizure disorder	generalised seizures	N	N		bilateral epileptiform activity
mohamad arshad	1yr	M	41668	no	myoclonic epilepsy	myoclonic seizures	delay		N	3hz spike and wave discharge
Thamanesh	1yr	M	35465	no	seizure disorder	generalised seizures	N		atrophic changes	bilateral epileptiform activity
maheshbala	2yrs	M	14165	no	Atypical febrile seizure	generalised seizures	N		n	bilateral epileptiform activity
sudeep	2yrs	M	39432	no	Atypical febrile seizure	generalised seizures	N		n	bilateral epileptiform activity
sooryamoorthy	2yrs	M	11981	no	Atypical febrile seizure	partial seizures	N		n	3hz spike wave discharge
Kanishka	2yrs	F	32920	no	Atypical febrile seizure	generalised seizures	N		N	N
Essakkiammal	2Yrs	F	35108	no	Atypical febrile seizure	generalised seizures	N		N	N
aruna devi	6yrs	F	6118	no	seizure	generalised seizures	N		N	N
Essakkiraj	9months	M	36786	no	Atypical febrile seizure	generalised seizures	N		N	N
Essakimayandi	2yrs	M	33543	no	Atypical febrile seizure	partial seizures	N		N	focal spike and wave discharge
Vignesh	2yrs	M	35116	no	Status epilepticus	partial seizures	N		Bl gliosis of parietal lobe. Demyelination	focal spike and wave discharge
Ajay	3months	M	34882	no	Seizure	generalised seizures	N		N	bilateral epileptiform activity
Sundarajan	3months	M	42876	no	Status epilepticus	generalised seizures	N		N	bilateral epileptiform activity
Jeswin	2yrs	M	33652	no	seizure disorder	partial seizures	N		N	N
esaiprakash	3yrs	M	42108	no	Atypical febrile seizure	generalised seizures	N		N	fronto central spikes

sasikumar	3yrs	M	49567	yes	Seizure	generalised seizures	N		N	focal spike and wave discharge
ragavendran	3yrs	M	11439	no	Tuberculoma	partial seizures	N	ring enhancing lesion	ring enhancing lesion	focal spike and wave discharge
Anand	4months	M	34543	yes	Seizure	generalised seizures	N	N		focal spike and wave discharge
Durga	4yrs	F	33617	yes	seizure disorder	generalised seizures	N		N	bilateral epileptiform activity
Nivisha	4yrs	F	42608	no	Seizure	generalised seizures	N		N	intermittentslowing and spike wave activity bl centroparietal region
Sunil	4yrs	M	44969	no	Atypical febrile seizure	generalised seizures	N		N	focal spike and wave discharge
Keerthika	4yrs	F	43399	no	status epilepticus	generalised seizures	N		N	bilateral epileptiform activity
guna	4yrs	M	48181	no	absence seizure	generalised seizures	N		N	3hz spike and wave discharge
banupriya	5yrs	F	42134	no	absence seizure	generalised seizures	N		N	3hz spike and wave discharge
sathish	5yrs	M	22779	yes	nocturnal seizures	generalised seizures	N		N	bilateral epileptiform activity
muthuraman	5yrs	M	37620	no	seizure disorder	generalised seizures	N		n	bilateral epileptiform activity
Anushya	6yrs	F	34513	yes	Seizure	generalised seizures	N	arnold chiari malformation		bilateral epileptiform activity
Amudha	2yrs	F	36660	yes	Atypical febrile seizure	partial seizures	N		N	N
Keronisha	3months	F	32665	no	Status epilepticus	partial seizures	N	Ac subdural bleed rt frontal convexity rt parieto occcopital convexity	Sih bifrontal region midline shift	N
Ramya	6yrs	F	45804	yes	seizure disorder	partial seizures	N		N	Background delta,focal sharp waves, focal spike waves,
chitruvan	7months	M	23765	no	Status epilepticus	generalised seizures	N		dilated 3rd ventricle	focal spike and wave discharge
karthikeyan	7yrs	M	20958	yes	seizure disorder	generalised seizures	N		n	bilateral epileptiform activity
saranya	7yrs	F	6061	no	seizure disorder	generalised seizures	N		N	focal spike and wave discharge
Vijayaraja	8yrs	M	42785	no	seizure disorder	generalised seizures	N	N		bilateral epileptiform activity
syed ali fathima	8yrs	F	15932	no	tuberous sclerosis	partial seizures	N	cortical tubers/ tuberous sclerosis	cortical tubers	bilateral epileptiform activity
rajesh	8yrs	M	31879	yes	nocturnal seizures	partial seizures	n	n		bilateral epileptiform activity
Satish	8yrs	M	36538	no	seizure disorder	partial seizures	N		White matter hypodensity temperoparital lobe	focal spike and wave discharge
abinesh	8yrs	M	28172	no	Status epilepticus	partial seizures	N		n	focal spike and wave discharge
Muthuselvi	6yrs	F	36973	no	Atypical febrile seizure	partial seizures	N		N	N
Aravaind	9yrs	M	39552	no	seizure disorder	generalised seizures	N	n		bilateral epileptiform activity